EAST Search History

Ref #	Hits	Search Query	DBs	Default Operator	Plurals	Time Stamp
L1	112	"5512549"	US-PGPUB; USPAT; USOCR; EPO; JPO; DERWENT	AND	ON	2007/01/27 11:54
S1	5	Hathaway.IN. Baron.IN. Mistry.IN. Roman.IN.	US-PGPUB; USPAT; USOCR; EPO; JPO; DERWENT	AND	ON	2007/01/27 11:53
S2	112	"5512549"	US-PGPUB; USPAT; USOCR; EPO; JPO; DERWENT	AND	ON	2007/01/26 19:06
S3	2	"20020107206"	US-PGPUB; USPAT; USOCR; EPO; JPO; DERWENT	AND	ON	2007/01/26 19:06

=> D Hist

(FILE 'HOME' ENTERED AT 15:33:15 ON 24 JAN 2007)

FILE 'BIOSIS, CAPLUS, EMBASE, MEDLINE' ENTERED AT 15:33:32 ON 24 JAN 2007
L1 9302 S EXENDIN OR GLP-1 OR ("GLUCAGON-LIKE AGONIST")
L2 24309 S "FREE RADICAL SCAVENGER"
L3 44707 S ISCHEM####### AND EVENT
L4 158841 S REPERFUSION
L5 183126 S CARDIAC INTERVENTION OR (ANGIOPLASTY OR "CORONARY BY PASS" OR
L6 110219 S CARDIAC AND (ISCHEMIA OR REPERFUSION OR "CONGESTIVE HEART FAI
L7 355 S METABOLIC INTERVENTION
L8 74695 S ARRHYTHMIA AND (TREAT##### OR PREVENT#######)
L9 4 S L1 AND L8 AND PD<=20031219
L10 1 S L1 AND L2 AND PD<=20031219
L11 0 S L1 AND L3 AND PD<=20031219
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L17 4 DUP REM L9 (0 DUPLICATES REMOVED)
L18 8 DUP REM L13 (7 DUPLICATES REMOVED)
L19 2 DUP REM L14 (0 DUPLICATES REMOVED)
L20 8 DUP REM L15 (0 DUPLICATES REMOVED)
L21 4 DUP REM L16 (1 DUPLICATE REMOVED)

=> S Nephropathy OR ("END Stage renal disease") OR ESRD
L22 139696 NEPHROPATHY OR ("END STAGE RENAL DISEASE") OR ESRD

=> S Endothelial Function
L23 22772 ENDOTHELIAL FUNCTION

=> S ProteinUria
L24 82699 PROTEINURIA

=> S Glomerulosclerosis
L25 21397 GLOMERULOSCLEROSIS

=> s Diabetes OR "Insulin resistance" OR hypertension
L26 1521989 DIABETES OR "INSULIN RESISTANCE" OR HYPERTENSION

=> S L1 AND L22 AND pd<=20031219
3 FILES SEARCHED...
L27 14 L1 AND L22 AND PD<=20031219

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3 FILES SEARCHED...
L30 1 L1 AND L25 AND PD<=20031219

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3 FILES SEARCHED...
L31 1792 L1 AND L26 AND PD<=20031219

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L32 13 L31 AND L22

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L8 74695 S ARRHYTHMIA AND (TREAT##### OR PREVENT#######)
L9 4 S L1 AND L8 AND PD<=20031219
L10 1 S L1 AND L2 AND PD<=20031219
L11 0 S L1 AND L3 AND PD<=20031219
L12 0 S L1 AND L3 AND PD<=20031219
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L17 4 DUP REM L9 (0 DUPLICATES REMOVED)
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L19 2 DUP REM L14 (0 DUPLICATES REMOVED)
L20 8 DUP REM L15 (0 DUPLICATES REMOVED)
L21 4 DUP REM L16 (1 DUPLICATE REMOVED)
L22 139696 S NEPHROPATHY OR ("END STAGE RENAL DISEASE") OR ESRD
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L27 14 S L1 AND L22 AND PD<=20031219
L28 1 S L1 AND L23 AND PD<=20031219
L29 1 S L1 AND L24 AND PD<=20031219
L30 1 S L1 AND L25 AND PD<=20031219
L31 1792 S L1 AND L26 AND PD<=20031219
L32 13 S L31 AND L22

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In a multifile environment, a format can only be used if it is valid in at least one of the files. Refer to file specific help messages or the STNGUIDE file for information on formats available in individual files.
REENTER DISPLAY FORMAT FOR ALL FILES (FILEDEFAULT):
REENTER DISPLAY FORMAT FOR ALL FILES (FILEDEFAULT):Exit
'EXIT' IS NOT A VALID FORMAT
In a multifile environment, a format can only be used if it is valid in at least one of the files. Refer to file specific help messages or the STNGUIDE file for information on formats available in individual files.
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'DUP' IS NOT A VALID FORMAT
'REM' IS NOT A VALID FORMAT
'L115' IS NOT A VALID FORMAT
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In a multifile environment, a format can only be used if it is valid in at least one of the files. Refer to file specific help messages or the STNGUIDE file for information on formats available in individual files.
REENTER DISPLAY FORMAT FOR ALL FILES (FILEDEFAULT):Ibib

L27 ANSWER 1 OF 14 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on STN
ACCESSION NUMBER: 2003:390202 BIOSIS <<LOGINID::20070124>>
DOCUMENT NUMBER: PREV200300390202
TITLE: The glucagon-like peptides: A double-edged therapeutic sword?.
AUTHOR(S): Perry, TracyAnn [Reprint Author]; Greig, Nigel H.
CORPORATE SOURCE: Section of Drug Design and Development, Laboratory of Neurosciences, Gerontology Research Center, National Institute on Aging, National Institutes of Health, 5600 Nathan Shock Drive, Baltimore, MD, 21224, USA perryt@grc.nia.nih.gov
SOURCE: Trends in Pharmacological Sciences, (July 2003) Vol. 24, No. 7, pp. 377-383. print. ISSN: 0165-6147 (ISSN print).
DOCUMENT TYPE: Article
General Review; (Literature Review)
LANGUAGE: English
ENTRY DATE: Entered STN: 27 Aug 2003
Last Updated on STN: 27 Aug 2003

=> Dup Rem l27
PROCESSING COMPLETED FOR L27
L33 9 DUP REM L27 (5 DUPLICATES REMOVED)

=> Dup Rem l32
PROCESSING COMPLETED FOR L32
L34 9 DUP REM L32 (4 DUPLICATES REMOVED)

=> D Ibib All L28

L28 ANSWER 1 OF 1 CAPLUS COPYRIGHT 2007 ACS on STN
ACCESSION NUMBER: 2003:447519 CAPLUS <<LOGINID::20070124>>
DOCUMENT NUMBER: 139:255595
TITLE: Antihypertensive effect of glucagon-like peptide 1 in Dahl salt-sensitive rats
AUTHOR(S): Yu, Ming; Moreno, Carol; Hoagland, Kimberly M.; Dahly, Annette; Ditter, Katie; Mistry, Mahesh; Roman, Richard J.
CORPORATE SOURCE: Department of Physiology, Medical College of Wisconsin, Milwaukee, WI, 53226, USA
SOURCE: Journal of Hypertension (2003), 21(6), 1125-1135
CODEN: JOHYD3; ISSN: 0263-6352
PUBLISHER: Lippincott Williams & Wilkins

DOCUMENT TYPE: Journal
LANGUAGE: English
AN 2003:447519 CAPLUS <<LOGINID::20070124>>
DN 139:255595
ED Entered STN: 11 Jun 2003
TI Antihypertensive effect of glucagon-like peptide 1 in Dahl salt-sensitive rats
AU Yu, Ming; Moreno, Carol; Hoagland, Kimberly M.; Dahly, Annette; Ditter, Katie; Mistry, Mahesh; Roman, Richard J.
CS Department of Physiology, Medical College of Wisconsin, Milwaukee, WI, 53226, USA
SO Journal of Hypertension (2003), 21(6), 1125-1135
CODEN: JOHYD3; ISSN: 0263-6352
PB Lippincott Williams & Wilkins
DT Journal
LA English
CC 2-6 (Mammalian Hormones)
AB Dahl salt-sensitive (Dahl S) rats exhibit many phenotypic traits associated with salt-sensitive hypertension in man. Specifically, they are salt-sensitive, insulin-resistant and hyperlipidemic. They also develop endothelial dysfunction, cardiac injury and glomerulosclerosis. Insulin resistance is linked to hypertension, renal and cardiac damage and endothelial dysfunction. Thus, an agent that has diuretic action and can improve insulin resistance, like recombinant glucagon-like peptide-1(7-36)amide (rGLP-1), may have an antihypertensive effect. To determine whether chronic administration of rGLP-1 attenuates the development of hypertension, endothelial dysfunction and/or hypertension-induced renal and cardiac end organ damage in Dahl S rats. Mean arterial pressure (MAP) and urinary excretion of protein and albumin were measured in Dahl S rats before and after they were fed a 8% NaCl diet and infused with rGLP-1 (1 mg/kg per min, i.v.) or vehicle for 14 days. At the end of the study, the degree of renal and cardiac injury was histol. assessed and endothelium-dependent relaxing function was studied using aortic rings. In other rats, the effects of rGLP-1 on sodium and water balance and plasma glucose and insulin levels for the first 3 days following a step change in sodium intake from a 0.1% NaCl diet to 7.5 mEq/day were determined The rGLP-1 significantly attenuated the development of hypertension in Dahl S rats (136 vs. 174 mmHg). This was associated with reduction in proteinuria (46 vs. 128 mg/day) and albuminuria (46 vs. 86 mg/day) and improvement of endothelial function and renal and cardiac damage. The rGLP-1 markedly increased urine flow and sodium excretion for the first 3 days following elevation in sodium intake. It had no significant effects on plasma glucose and insulin concns. The rGLP-1 has antihypertensive and cardiac and renoprotective effects in Dahl S rats fed a high salt diet. The antihypertensive effect of rGLP-1 in Dahl S rats is due mainly to its diuretic and natriuretic effects, rather than an effect to improve insulin-resistance.
ST hypertension GLP1; sodium water excretion hypertension GLP1; glucose insulin blood hypertension GLP1; aorta heart kidney damage hypertension GLP1; albuminuria proteinuria hypertension GLP1; antihypertensive GLP1 diuresis natriuresis
IT Hypertension
(Dahl salt-sensitive; antihypertensive effect of glucagon-like peptide 1 in Dahl salt-sensitive rats)
IT Heart, disease
Kidney, disease
(GLP-1 effect on aorta endothelium and heart and kidney damage in Dahl salt-sensitive hypertensive rats)
IT Blood pressure
Heart rate
(GLP-1 effect on blood pressure and heart rate in Dahl salt-sensitive hypertensive rats)
IT Albumins, biological studies
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(albuminuria; GLP-1 effect on albuminuria and proteinuria in Dahl salt-sensitive hypertensive rats)
IT Antihypertensives
(antihypertensive action of GLP-1 in Dahl salt-sensitive hypertensive rats is due to diuretic and natriuretic actions)
IT Artery, disease
(aortic endothelial injury; GLP-1 effect on aorta endothelium and heart and kidney damage in Dahl salt-sensitive hypertensive rats)
IT Injury
(aortic endothelial; GLP-1 effect on aorta endothelium and heart and kidney damage in Dahl salt-sensitive hypertensive rats)
IT Endothelium
(aortic, disease, injury; GLP-1 effect on aorta endothelium and heart and kidney damage in Dahl salt-sensitive hypertensive rats)
IT Proteins
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(proteinuria; GLP-1 effect on albuminuria and proteinuria in Dahl salt-sensitive hypertensive rats)
IT 50-99-7, Glucose, biological studies 9004-10-8, Insulin, biological studies
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(GLP-1 effect on blood glucose and insulin in Dahl salt-sensitive hypertensive rats)
IT 7440-23-5, Sodium, biological studies 7732-18-5, Water, biological studies
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(GLP-1 effect on water and sodium excretion in Dahl salt-sensitive hypertensive rats)
IT 89750-14-1, Glucagon-like peptide 1 118549-37-4, Insulinotropin
RL: BSU (Biological study, unclassified); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(antihypertensive effect of glucagon-like peptide 1 in Dahl salt-sensitive rats)
RE.CNT 44 THERE ARE 44 CITED REFERENCES AVAILABLE FOR THIS RECORD
RE

(1) Anderson, S; J Clin Invest 1985, V76, P612 CAPLUS
(2) Barragan, J; Am J Physiol 1994, V266, PE459 CAPLUS
(3) Bishop, J; Cardiovasc Res 2000, V47, P57 CAPLUS
(4) Bullock, B; Endocrinology 1996, V137, P2968 CAPLUS
(5) Campese, V; Hypertension 1994, V23, P531 MEDLINE
(6) Dall'Aglio, E; Am J Hypertens 1991, V4, P773 CAPLUS
(7) DeFronzo, R; Diabetologia 1981, V21, P165 CAPLUS
(8) DeFronzo, R; J Clin Invest 1976, V58, P83 CAPLUS
(9) Dengel, D; Hypertension 1996, V28, P127 MEDLINE
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(14) Hayakawa, H; Circulation 1997, V96, P2407 CAPLUS
(15) Ito, O; Hypertension 1999, V33, P419 CAPLUS
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(20) Miller, A; J Cardiovasc Pharm Ther 1998, V3, P125 CAPLUS
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(30) Rapp, J; Hypertension 1982, V4, P753 MEDLINE
(31) Reaven, G; Hypertension 1991, V18, P630 CAPLUS
(32) Ritz, E; J Intern Med 1999, V245, P111 MEDLINE
(33) Roman, R; Am J Hypertens 1997, V10, P63S CAPLUS
(34) Sakamoto, S; Diabetes 1998, V47, P82 CAPLUS
(35) Salonen, J; Diabetes 1998, V47, P270 CAPLUS
(36) Shimabukuro, M; Metabolism 1996, V45, P1168 CAPLUS
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(41) Vella, A; Diabetes 2000, V49, P611 CAPLUS
(42) Yagi, K; Hypertension 1997, V29, P728 CAPLUS
(43) Yamamoto, H; J Clin Invest 2002, V110, P43 CAPLUS
(44) Zou, A; Hypertension 1996, V27, P631 CAPLUS

=> D Ibib ALL l28

L28 ANSWER 1 OF 1 CAPLUS COPYRIGHT 2007 ACS on STN
ACCESSION NUMBER: 2003:447519 CAPLUS <<LOGINID::20070124>>
DOCUMENT NUMBER: 139:255595
TITLE: Antihypertensive effect of glucagon-like peptide 1 in Dahl salt-sensitive rats
AUTHOR(S): Yu, Ming; Moreno, Carol; Hoagland, Kimberly M.; Dahly, Annette; Ditter, Katie; Mistry, Mahesh; Roman, Richard J.
CORPORATE SOURCE: Department of Physiology, Medical College of Wisconsin, Milwaukee, WI, 53226, USA
SOURCE: Journal of Hypertension (2003), 21(6), 1125-1135
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PUBLISHER: Lippincott Williams & Wilkins
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LANGUAGE: English
AN 2003:447519 CAPLUS <<LOGINID::20070124>>
DN 139:255595
ED Entered STN: 11 Jun 2003
TI Antihypertensive effect of glucagon-like peptide 1 in Dahl salt-sensitive rats
AU Yu, Ming; Moreno, Carol; Hoagland, Kimberly M.; Dahly, Annette; Ditter, Katie; Mistry, Mahesh; Roman, Richard J.
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CODEN: JOHYD3; ISSN: 0263-6352
PB Lippincott Williams & Wilkins
DT Journal
LA English
CC 2-6 (Mammalian Hormones)
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with salt-sensitive hypertension in man. Specifically, they are salt-sensitive, insulin-resistant and hyperlipidemic. They also develop endothelial dysfunction, cardiac injury and glomerulosclerosis. Insulin resistance is linked to hypertension, renal and cardiac damage and endothelial dysfunction. Thus, an agent that has diuretic action and can improve insulin resistance, like recombinant glucagon-like peptide-1(7-36)amide (rGLP-1), may have an antihypertensive effect. To determine whether chronic administration of rGLP-1 attenuates the development of hypertension, endothelial dysfunction and/or hypertension-induced renal and cardiac end organ damage in Dahl S rats. Mean arterial pressure (MAP) and urinary excretion of protein and albumin were measured in Dahl S rats before and after they were fed a 8% NaCl diet and infused with rGLP-1 (1 mg/kg per min, i.v.) or vehicle for 14 days. At the end of the study, the degree of renal and cardiac injury was histol. assessed and endothelium-dependent relaxing function was studied using aortic rings. In other rats, the effects of rGLP-1 on sodium and water balance and plasma glucose and insulin levels for the first 3 days following a step change in sodium intake from a 0.1% NaCl diet to 7.5 mEq/day were determined The rGLP-1 significantly attenuated the development of hypertension in Dahl S rats (136 vs. 174 mmHg). This was associated with reduction in proteinuria (46 vs. 128 mg/day) and albuminuria (46 vs. 86 mg/day) and improvement of endothelial function and renal and cardiac damage. The rGLP-1 markedly increased urine flow and sodium excretion for the first 3 days following elevation in sodium intake. It had no significant effects on plasma glucose and insulin concns. The rGLP-1 has antihypertensive and cardiac and renoprotective effects in Dahl S rats fed a high salt diet. The antihypertensive effect of rGLP-1 in Dahl S rats is due mainly to its diuretic and natriuretic effects, rather than an effect to improve insulin-resistance.

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IT Hypertension
(Dahl salt-sensitive; antihypertensive effect of glucagon-like peptide 1 in Dahl salt-sensitive rats)

IT Heart, disease
Kidney, disease
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IT Blood pressure
Heart rate
(GLP-1 effect on blood pressure and heart rate in Dahl salt-sensitive hypertensive rats)

IT Albumins, biological studies
RL: BSU (Biological study, unclassified); BIOL (Biological study)
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(34) Sakamoto, S; Diabetes 1998, V47, P82 CAPLUS
(35) Salonen, J; Diabetes 1998, V47, P270 CAPLUS
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=> D fbib all l29

L29 ANSWER 1 OF 1 CAPLUS COPYRIGHT 2007 ACS on STN
ACCESSION NUMBER: 2003:447519 CAPLUS <<LOGINID::20070124>>
DOCUMENT NUMBER: 139:255595
TITLE: Antihypertensive effect of glucagon-like peptide 1 in Dahl salt-sensitive rats

AUTHOR(S): Yu, Ming; Moreno, Carol; Hoagland, Kimberly M.; Dahly, Annette; Ditter, Katie; Mistry, Mahesh; Roman, Richard J.
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LA English

CC 2-6 (Mammalian Hormones)

AB Dahl salt-sensitive (Dahl S) rats exhibit many phenotypic traits associated with salt-sensitive hypertension in man. Specifically, they are salt-sensitive, insulin-resistant and hyperlipidemic. They also develop endothelial dysfunction, cardiac injury and glomerulosclerosis. Insulin resistance is linked to hypertension, renal and cardiac damage and endothelial dysfunction. Thus, an agent that has diuretic action and can improve insulin resistance, like recombinant glucagon-like peptide-1(7-36)amide (rGLP-1), may have an antihypertensive effect. To determine whether chronic administration of rGLP-1 attenuates the development of hypertension, endothelial dysfunction and/or hypertension-induced renal and cardiac end organ damage in Dahl S rats. Mean arterial pressure (MAP) and urinary excretion of protein and albumin were measured in Dahl S rats before and after they were fed a 8% NaCl diet and infused with rGLP-1 (1 mg/kg per min, i.v.) or vehicle for 14 days. At the end of the study, the degree of renal and cardiac injury was histol. assessed and endothelium-dependent relaxing function was studied using aortic rings. In other rats, the effects of rGLP-1 on sodium and water balance and plasma glucose and insulin levels for the first 3 days following a step change in sodium intake from a 0.1% NaCl diet to 7.5 mEq/day were determined

The rGLP-1 significantly attenuated the development of hypertension in Dahl S rats (136 vs. 174 mmHg). This was associated with reduction in proteinuria (46 vs. 128 mg/day) and albuminuria (46 vs. 86 mg/day) and improvement of endothelial function and renal and cardiac damage. The rGLP-1 markedly increased urine flow and sodium excretion for the first 3 days following elevation in sodium intake. It had no significant effects on plasma glucose and insulin concns. The rGLP-1 has antihypertensive and cardiac and renoprotective effects in Dahl S rats fed a high salt diet. The antihypertensive effect of rGLP-1 in Dahl S rats is due mainly to its diuretic and natriuretic effects, rather than an effect to improve insulin-resistance.

ST hypertension GLP1; sodium water excretion hypertension GLP1; glucose insulin blood hypertension GLP1; aorta heart kidney damage hypertension GLP1; albuminuria proteinuria hypertension GLP1; antihypertensive GLP1 diuresis natriuresis

IT Hypertension
(Dahl salt-sensitive; antihypertensive effect of glucagon-like peptide 1 in Dahl salt-sensitive rats)

IT Heart, disease
Kidney, disease
(GLP-1 effect on aorta endothelium and heart and kidney damage in Dahl salt-sensitive hypertensive rats)

IT Blood pressure
Heart rate
(GLP-1 effect on blood pressure and heart rate in Dahl salt-sensitive hypertensive rats)

IT Albumins, biological studies
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(albuminuria; GLP-1 effect on albuminuria and proteinuria in Dahl salt-sensitive hypertensive rats)

IT Antihypertensives
(antihypertensive action of GLP-1 in Dahl salt-sensitive hypertensive rats is due to diuretic and natriuretic actions)

IT Artery, disease
(aortic endothelial injury; GLP-1 effect on aorta endothelium and heart and kidney damage in Dahl salt-sensitive hypertensive rats)

IT Injury
(aortic endothelial; GLP-1 effect on aorta endothelium and heart and kidney damage in Dahl salt-sensitive hypertensive rats)

IT Endothelium
(aortic, disease, injury; GLP-1 effect on aorta endothelium and heart and kidney damage in Dahl salt-sensitive hypertensive rats)

IT Proteins
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(proteinuria; GLP-1 effect on albuminuria and proteinuria in Dahl salt-sensitive hypertensive rats)

IT 50-99-7, Glucose, biological studies 9004-10-8, Insulin, biological studies
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(GLP-1 effect on blood glucose and insulin in Dahl salt-sensitive hypertensive rats)

IT 7440-23-5, Sodium, biological studies 7732-18-5, Water, biological studies
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(GLP-1 effect on water and sodium excretion in Dahl salt-sensitive hypertensive rats)

IT 89750-14-1, Glucagon-like peptide I 118549-37-4, Insulinotropin
RL: BSU (Biological study, unclassified); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(antihypertensive effect of glucagon-like peptide 1 in Dahl salt-sensitive rats)

RE.CNT 44 THERE ARE 44 CITED REFERENCES AVAILABLE FOR THIS RECORD

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=> D Ibib l30

L30 ANSWER 1 OF 1 CAPLUS COPYRIGHT 2007 ACS on STN
ACCESSION NUMBER: 2003:447519 CAPLUS <<LOGINID::20070124>>
DOCUMENT NUMBER: 139:255595
TITLE: Antihypertensive effect of glucagon-like peptide 1 in Dahl salt-sensitive rats
AUTHOR(S): Yu, Ming; Moreno, Carol; Hoagland, Kimberly M.; Dahly, Annette; Ditter, Katie; Mistry, Mahesh; Roman, Richard J.
CORPORATE SOURCE: Department of Physiology, Medical College of Wisconsin, Milwaukee, WI, 53226, USA
SOURCE: Journal of Hypertension (2003), 21(6), 1125-1135
CODEN: JOHYD3; ISSN: 0263-6352
PUBLISHER: Lippincott Williams & Wilkins
DOCUMENT TYPE: Journal
LANGUAGE: English
REFERENCE COUNT: 44 THERE ARE 44 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

=> D Ibib All L33 1-9

L33 ANSWER 1 OF 9 CAPLUS COPYRIGHT 2007 ACS on STN
ACCESSION NUMBER: 2004:533962 CAPLUS <<LOGINID::20070124>>
DOCUMENT NUMBER: 141:82335
TITLE: Human glucagon-like-peptide-1 mimics and their antidiabetic effects
INVENTOR(S): Natarajan, Sesha Iyer; Mapelli, Claudio; Bastos, Margarita M.; Bernatowicz, Michael; Lee, Ving; Ewing, William R.
PATENT ASSIGNEE(S): USA
SOURCE: U.S. Pat. Appl. Publ., 73 pp., Cont.-in-part of U.S. Ser. No. 273,975.
CODEN: USXXCO
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 2
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
US 2004127423	A1	20040701	US 2003-419399	20030421
US 2003195157	A1	20031016	US 2002-273975	20021018 <--
WO 2004094461	A2	20041104	WO 2004-US12374	20040421
WO 2004094461	A3	20050915		

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW
RW: BW, GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG

EP 1615653	A2	20060118	EP 2004-760098	20040421

R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR, BG, CZ, EE, HU, PL, SK, HR

PRIORITY APPLN. INFO.: US 2001-342015P P 20011018
US 2002-273975 A2 20021018
US 2003-419399 A 20030421
WO 2004-US12374 W 20040421

AN 2004:533962 CAPLUS <<LOGINID::20070124>>
DN 141:82335
ED Entered STN: 02 Jul 2004
TI Human glucagon-like-peptide-1 mimics and their antidiabetic effects
IN Natarajan, Sesha Iyer; Mapelli, Claudio; Bastos, Margarita M.;

Bernatowicz, Michael; Lee, Ving; Ewing, William R.
PA USA
SO U.S. Pat. Appl. Publ., 73 pp., Cont.-in-part of U.S. Ser. No. 273,975.
CODEN: USXXCO
DT Patent
LA English
IC ICM A61K038-10
ICS C07K007-08
INCL 514015000; 530328000
CC 1-10 (Pharmacology)
Section cross-reference(s): 2, 34, 63
FAN.CNT 2

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI US 2004127423	A1	20040701	US 2003-419399	20030421
US 2003195157	A1	20031016	US 2002-273975	20021018 <--
WO 2004094461	A2	20041104	WO 2004-US12374	20040421
WO 2004094461	A3	20050915		

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW
RW: BW, GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG

EP 1615653	A2	20060118	EP 2004-760098	20040421

R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR, BG, CZ, EE, HU, PL, SK, HR

PRAI US 2001-342015P P 20011018
US 2002-273975 A2 20021018
US 2003-419399 A 20030421
WO 2004-US12374 W 20040421
CLASS

PATENT NO.	CLASS	PATENT FAMILY CLASSIFICATION CODES
US 2004127423	ICM	A61K038-10
	ICS	C07K007-08
	INCL	514015000; 530328000
	IPCI	A61K0038-10 [ICM,7]; C07K0007-08 [ICS,7]; C07K0007-00 [ICS,7,C*]
	IPCR	A61K0038-00 [N,C*]; A61K0038-00 [N,A]; C07K0014-435 [I,C*]; C07K0014-605 [I,A]
	NCL	514/015.000; 530/328.000
	ECLA	C07K014/605
US 2003195157	IPCI	A61K0038-10 [ICM,7]; A61K0038-08 [ICS,7]; C07K0007-08 [ICS,7]; C07K0007-06 [ICS,7]; C07K0007-00 [ICS,7,C*]
	IPCR	A61K0038-00 [N,C*]; A61K0038-00 [N,A]; C07K0014-435 [I,C*]; C07K0014-605 [I,A]
	NCL	514/016.000; 514/017.000; 530/328.000; 530/329.000
	ECLA	C07K014/605
WO 2004094461	IPCI	C07K [ICM,7]
	IPCR	A61K0038-00 [I,C*]; A61K0038-00 [I,A]; A61K0038-02 [I,C*]; A61K0038-02 [I,A]; A61K0038-08 [I,C*]; A61K0038-08 [I,A]; A61K0038-10 [I,C*]; A61K0038-10 [I,A]; C07K [I,S]; C07K0007-00 [I,C*]; C07K0007-02 [I,A]; C07K0007-04 [I,A]; C07K0007-08 [I,A]
EP 1615653	IPCI	A61K0038-00 [ICM,7]; A61K0038-02 [ICS,7]; A61K0038-10 [ICS,7]; A61K0038-08 [ICS,7]; C07K0007-02 [ICS,7]; C07K0007-04 [ICS,7]; C07K0007-08 [ICS,7]; C07K0007-00 [ICS,7,C*]
	IPCR	A61K0038-00 [I,C*]; A61K0038-00 [I,A]; A61K0038-02 [I,C*]; A61K0038-02 [I,A]; A61K0038-08 [I,C*]; A61K0038-08 [I,A]; A61K0038-10 [I,C*]; A61K0038-10 [I,A]; C07K [I,S]; C07K0007-00 [I,C*]; C07K0007-02 [I,A]; C07K0007-04 [I,A]; C07K0007-08 [I,A]

AB The invention discloses human glucagon-like peptide-1 (GLP-1) peptide mimics that mimic the biol. activity of the native GLP-1 peptide and thus are useful for the treatment or prevention of diseases or disorders associated with GLP activity. Further, the invention provides novel, chemical modified peptides that not only stimulate insulin secretion in type II diabetics, but also produce other beneficial insulinotropic responses. These synthetic peptide GLP -1 mimics exhibit increased stability to proteolytic cleavage making them ideal therapeutic candidates for oral or parenteral administration.

ST human glucagon peptide mimic prepn diabetes antidiabetic insulin stability

IT Proteins
RL: PAC (Pharmacological activity); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(ALBP (adipocyte lipid-binding protein); human glucagon-like-peptide-1 mimics and their antidiabetic effects)

IT Lipoprotein receptors
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(LDL; human glucagon-like-peptide-1 mimics and their antidiabetic effects)

IT Proteins
RL: PAC (Pharmacological activity); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(MTP (microsomal triglyceride-exchanging protein), inhibitors; human glucagon-like-peptide-1 mimics and their antidiabetic effects)

IT Antiarteriosclerotics
(antiatherosclerotics; human glucagon-like-peptide-1 mimics and their antidiabetic effects)

IT Drug delivery systems
(capsules; human glucagon-like-peptide-1 mimics and their antidiabetic effects)

IT Proteins
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(cholesterol ester-exchanging; human glucagon-like-peptide-1 mimics and their antidiabetic effects)

IT Kidney, disease
(diabetic nephropathy; human glucagon-like-peptide-1 mimics and their antidiabetic effects)

IT Nerve, disease
(diabetic neuropathy; human glucagon-like-peptide-1 mimics and their antidiabetic effects)

IT Eye, disease
(diabetic retinopathy; human glucagon-like-peptide-1 mimics and their antidiabetic effects)

IT Transport proteins
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(dopamine transporter; human glucagon-like-peptide-1 mimics and their antidiabetic effects)

IT 5-HT reuptake inhibitors
Antihypertensives
Antiobesity agents
Appetite depressants
Atherosclerosis
Diabetes mellitus
Human
Hyperglycemia
Hypertension
Hypertriglyceridemia
Hypolipemic agents
Obesity
Signal transduction, biological
Wound healing
b3-Adrenoceptor agonists
(human glucagon-like-peptide-1 mimics and their antidiabetic effects)

IT Fatty acids, biological studies
Glucagon-like peptide-1 receptors
Hyperlipidemia
Thyroid hormone receptors
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(human glucagon-like-peptide-1 mimics and their antidiabetic effects)

IT Peptides, biological studies
RL: PAC (Pharmacological activity); SPN (Synthetic preparation); THU (Therapeutic use); BIOL (Biological study); PREP (Preparation); USES (Uses)
(human glucagon-like-peptide-1 mimics and their antidiabetic effects)

IT Sulfonylureas
RL: PAC (Pharmacological activity); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(human glucagon-like-peptide-1 mimics and their antidiabetic effects)

IT Drug delivery systems
(injections; human glucagon-like-peptide-1 mimics and their antidiabetic effects)

IT Metabolic disorders
(metabolic syndrome X; human glucagon-like-peptide-1 mimics and their antidiabetic effects)

IT Drug delivery systems
(microparticles; human glucagon-like-peptide-1 mimics and their antidiabetic effects)

IT Diabetes mellitus
(non-insulin-dependent; human glucagon-like-peptide-1 mimics and their antidiabetic effects)

IT Antidiabetic agents
Drug delivery systems
(oral; human glucagon-like-peptide-1 mimics and their antidiabetic effects)

IT Drug delivery systems
(suspensions; human glucagon-like-peptide-1 mimics and their antidiabetic effects)

IT Drug delivery systems
(tablets; human glucagon-like-peptide-1 mimics and their antidiabetic effects)

IT Peroxisome proliferator-activated receptors
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(a; human glucagon-like-peptide-1 mimics and their antidiabetic effects)

IT Peroxisome proliferator-activated receptors
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(g; human glucagon-like-peptide-1 mimics and their antidiabetic effects)

IT 51-61-6, Dopamine, biological studies 56-81-5, Glycerol, biological studies 9001-62-1, Lipase 9028-35-7, 3-Hydroxy-3-methylglutaryl CoA reductase 9029-60-1, Lipoxygenase 9033-06-1, Glucosidase 9077-14-9, Squalene synthetase 63551-74-6, Lipoxygenase 90002-36-1, 2-Ethylphenyl boronic acid
RL: BSU (Biological study, unclassified); BIOL (Biological study)

(human glucagon-like-peptide-1 mimics and their antidiabetic effects)
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516521-54-3P 516521-55-4P 713497-71-3P 713497-72-4P 713497-73-5P
713497-74-6P 713497-75-7P 713497-77-9P
RL: PAC (Pharmacological activity); PRP (Properties); SPN (Synthetic preparation); THU (Therapeutic use); BIOL (Biological study); PREP (Preparation); USES (Uses)
(human glucagon-like-peptide-1 mimics and their antidiabetic effects)
IT 713497-79-1P 713497-81-5P 713497-83-7P 713497-85-9P
RL: PAC (Pharmacological activity); PRP (Properties); SPN (Synthetic preparation); THU (Therapeutic use); BIOL (Biological study); PREP (Preparation); USES (Uses)
(human glucagon-like-peptide-1 mimics and their antidiabetic effects)
IT 51-64-9, Dexamphet-amine 56-03-1, Biguanide 94-20-2, Chloropropamide 122-09-8, Phentermine 637-07-0, Clofibrate 657-24-9, Metformin 943-45-3D, Fibric acid, derivs. 10238-21-8, Glyburide 14838-15-4, Phenylpropanolamine 21187-98-4, Gliclazide 22232-71-9, Mazindol 25812-30-0, Gemfibrozil 29094-61-9, Glipizide 49562-28-9, Fenofibrate 54870-28-9, Meglitinide 56180-94-0, Acarbose 72432-03-2, Miglitol 75330-75-5, Lovastatin 79902-63-9, Simvastatin 81093-37-0, Pravastatin 89750-14-1, Glucagon-like peptide I 93479-97-1, Glimepiride 93957-54-1, Fluvastatin 96829-58-2, Orlistat 97240-79-4, Topiramate 97322-87-7, Troglitazone 105816-04-4, Nateglinide 106650-56-0, Sibutramine 111025-46-8, Pioglitazone 122320-73-4, Rosiglitazone 134523-00-5, Ator-vastatin 135062-02-1, Repaglinide 141750-63-2, Nisvastatin 141758-74-9, AC2993 144288-97-1, TS-962 145599-86-6, Cerivastatin 152755-31-2, LY295427 159183-92-3, L750355 161600-01-7, Isaglitazone 166518-60-1, Avasimibe 170861-63-9, JTT-501 176435-10-2, LY315902 178759-95-0, MD 700 196808-45-4 199113-98-9, NN-2344 199914-96-0, YM-440 213252-19-8, KRP297 244081-42-3, AJ9677 258345-41-4, GW-409544 262352-17-0, CP 529414 282526-98-1, ATL-962 287714-41-4 335149-08-1, L895645 335149-14-9, R-119702 335149-15-0, KAD1129 335149-17-2, AR-HO39242 335149-23-0, NVP-DPP-728A 335149-25-2, CP331648 430433-17-3, Glipyride 444069-80-1, Axokine
RL: PAC (Pharmacological activity); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(human glucagon-like-peptide-1 mimics and their antidiabetic effects)
IT 150-46-9, Triethylborate 358-23-6, Triflic anhydride 1973-22-4, 1-Bromo-2-ethylbenzene 4326-36-7 16419-60-6, O-Tolylboronic acid 82911-69-1 93267-04-0 516521-49-6 713497-86-0
RL: RCT (Reactant); RACT (Reactant or reagent)
(human glucagon-like-peptide-1 mimics and their antidiabetic effects)
IT 112766-18-4P 516521-46-3P 516521-47-4P 516521-48-5P 516521-50-9P 516521-51-0P
RL: RCT (Reactant); SPN (Synthetic preparation); PREP (Preparation); RACT (Reactant or reagent)
(human glucagon-like-peptide-1 mimics and their antidiabetic effects)
IT 713497-87-1P 713497-88-2P
RL: SPN (Synthetic preparation); PREP (Preparation)
(human glucagon-like-peptide-1 mimics and their antidiabetic effects)
IT 9027-63-8, ACAT
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(inhibitors; human glucagon-like-peptide-1 mimics and their antidiabetic effects)
IT 54249-88-6, Dipeptidyl peptidase IV
RL: PAC (Pharmacological activity); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(inhibitors; human glucagon-like-peptide-1 mimics and their antidiabetic effects)
IT 9004-10-8, Insulin, biological studies
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(resistance, hyperinsulinemia; human glucagon-like-peptide-1 mimics and their antidiabetic effects)

L33 ANSWER 2 OF 9 CAPLUS COPYRIGHT 2007 ACS on STN
ACCESSION NUMBER: 2004:157498 CAPLUS <<LOGINID::20070124>>
DOCUMENT NUMBER: 140:199313
TITLE: Preparation of fused pyrrolylcarboxamides as glycogen phosphorylase inhibitors
INVENTOR(S): Daisy, Joe
PATENT ASSIGNEE(S): Pfizer Products Inc., USA
SOURCE: Eur. Pat. Appl., 71 pp.
CODEN: EPXXDW
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 2
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
EP 1391460	A1	20040225	EP 2003-20676	20000918
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, FI, CY				
EP 1088824	A2	20010404	EP 2000-308131	20000918 <--
EP 1088824	A3	20010627		
EP 1088824	B1	20040107		
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO				
US 2002183369	A1	20021205	US 2002-117370	20020405 <--
US 6576653	B2	20030610		
US 2003195361	A1	20031016	US 2003-367002	20030214 <--
US 6828343	B2	20041207		

PRIORITY APPLN. INFO.: US 1999-157148P P 19990930
EP 2000-308131 A3 20000918
US 2000-670759 A3 20000927
US 2002-117370 A3 20020405
OTHER SOURCE(S): MARPAT 140:199313
AN 2004:157498 CAPLUS <<LOGINID::20070124>>
DN 140:199313
ED Entered STN: 26 Feb 2004
TI Preparation of fused pyrrolylcarboxamides as glycogen phosphorylase inhibitors
IN Daisy, Joe
PA Pfizer Products Inc., USA
SO Eur. Pat. Appl., 71 pp.
CODEN: EPXXDW
DT Patent
LA English
IC ICM C07D495-04
ICS C07D491-04; C07D209-52; A61K031-407; A61P003-10; A61P009-10; C07D495-14; C07D333-00; C07D209-00; C07D307-00
CC 28-2 (Heterocyclic Compounds (More Than One Hetero Atom))
Section cross-reference(s): 1, 63
FAN.CNT 2

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI EP 1391460	A1	20040225	EP 2003-20676	20000918
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, FI, CY				
EP 1088824	A2	20010404	EP 2000-308131	20000918 <--
EP 1088824	A3	20010627		
EP 1088824	B1	20040107		
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US 6576653	B2	20030610		
US 2003195361	A1	20031016	US 2003-367002	20030214 <--
US 6828343	B2	20041207		

PRAI US 1999-157148P P 19990930
EP 2000-308131 A3 20000918
US 2000-670759 A3 20000927
US 2002-117370 A3 20020405
CLASS

PATENT NO.	CLASS	PATENT FAMILY CLASSIFICATION CODES
EP 1391460	ICM	C07D495-04
	ICS	C07D491-04; C07D209-52; A61K031-407; A61P003-10; A61P009-10; C07D495-14; C07D333-00; C07D209-00; C07D307-00
	IPCI	C07D0495-04 [ICM,7]; C07D0491-04 [ICS,7]; C07D0491-00

[ICS,7,C*]; C07D0209-52 [ICS,7]; A61K0031-407 [ICS,7];
A61P0003-10 [ICS,7]; A61P0003-00 [ICS,7,C*];
A61P0009-10 [ICS,7]; A61P0009-00 [ICS,7,C*];
C07D0495-14 [ICS,7]; C07D0495-00 [ICS,7,C*];
C07D0333-00 [ICS,7]; C07D0209-00 [ICS,7]; C07D0307-00
[ICS,7]
ECLA C07D491/04+307B+209B; C07D495/04+333B+209B;
C07D495/14+333B+333B+209B
EP 1088824 IPCI C07D0495-04 [ICM,6]; C07D0491-04 [ICS,6]; C07D0209-52
[ICS,6]; A61K0031-407 [ICS,6]; A61P0003-10 [ICS,6];
A61P0003-00 [ICS,6,C*]; A61P0009-10 [ICS,6];
A61P0009-00 [ICS,6,C*]; C07D0495-04 [ICI,6];
C07D0495-00 [ICI,6,C*]; C07D0333-00 [ICI,6];
C07D0209-00 [ICI,6]; C07D0491-04 [ICI,6]; C07D0491-00
[ICI,6,C*]; C07D0307-00 [ICI,6]; C07D0209-00 [ICI,6]
IPCR C07D0491-048 [I,A]; A61K0031-407 [I,C*]; A61K0031-407
[I,A]; A61K0031-427 [I,C*]; A61K0031-427 [I,A];
A61K0031-4523 [I,C*]; A61K0031-454 [I,A]; A61K0031-5375
[I,C*]; A61K0031-5377 [I,A]; A61K0031-695 [I,C*];
A61K0031-695 [I,A]; A61K0038-00 [I,C*]; A61K0038-00
[I,A]; A61K0045-00 [I,C*]; A61K0045-00 [I,A];
A61P0003-00 [I,C*]; A61P0003-06 [I,A]; A61P0003-10
[I,A]; A61P0009-00 [I,C*]; A61P0009-10 [I,A];
A61P0009-12 [I,A]; A61P0027-00 [I,C*]; A61P0027-12
[I,A]; A61P0043-00 [I,C*]; A61P0043-00 [I,A];
C07D0209-00 [I,C*]; C07D0209-52 [I,A]; C07D0491-00
[I,C*]; C07D0491-04 [I,A]; C07D0495-00 [I,C*];
C07D0495-04 [I,A]; C07D0495-14 [I,A]; C07F0007-00
[I,C*]; C07F0007-10 [I,A]; C07K0005-00 [I,C*];
C07K0005-00 [I,A]
ECLA C07D209/52; C07D491/04+307B+209B; C07D495/04+333B+209B
US 2002183369 IPCI C07D0513-22 [ICM,7]; C07D0513-00 [ICM,7,C*];
A61K0031-429 [ICS,7]; A61K0031-424 [ICS,7];
A61K0031-4188 [ICS,7]; A61K0031-4164 [ICS,7,C*]
IPCR C07D0209-00 [I,C*]; C07D0209-52 [I,A]; C07D0491-00
[I,C*]; C07D0491-04 [I,A]; C07D0495-00 [I,C*];
C07D0495-04 [I,A]
NCL 514/367.000; 514/375.000; 514/393.000; 514/412.000;
548/153.000; 548/217.000; 548/303.100; 548/453.000
ECLA C07D209/52; C07D491/04+307B+209B; C07D495/04+333B+209B
US 2003195361 IPCI C07D0513-12 [ICM,7]; C07D0498-02 [ICS,7]; C07D0498-00
[ICS,7,C*]; C07D0513-02 [ICS,7]; C07D0513-00
[ICS,7,C*]; C07D0487-02 [ICS,7]; C07D0487-00 [ICS,7,C*]
IPCR C07D0209-00 [I,C*]; C07D0209-52 [I,A]; C07D0491-00
[I,C*]; C07D0491-04 [I,A]; C07D0495-00 [I,C*];
C07D0495-04 [I,A]
NCL 548/153.000; 548/218.000; 548/303.100; 548/453.000
ECLA C07D209/52; C07D491/04+307B+209B; C07D495/04+333B+209B
OS MARPAT 140:199313
GI

AB Title compds. [I; Q = substituted aryl, heteroaryl; Z, X = C, CH, CH2, N, O, S; X1 = NRa, CH2, O , S; dotted lines = bond, null; both dotted lines are not simultaneously bonds; R1 = H, halo, alkoxy, alkylthio, alkyl, CF3, NH2, alkylamino, dialkylamino, NO2, CN, CO2H, carboxyalkyl, alkenyl, alkynyl; Ra, Rb = H, alkyl; Y = CH(OH), null; R2R3 = atoms to form a 5-6 membered ring containing 0-3 heteroatoms and 0-2 double bonds; R4 = COA; A = NRdRd, NRaCH2CH2ORa, N-heterocyclyl; Rd = H, alkyl, alkoxy, aryl, (substituted) aryl, heteroaryl; Rc = H, CO2Ra, ORa, SRa, NRaRa; n = 1-3], were prepared for treatment of diabetes, insulin resistance, diabetic neuropathy, diabetic nephropathy, diabetic retinopathy, cataracts, hyperglycemia, hypercholesterolemia, hypertension, hyperinsulinemia, hyperlipidemia, atherosclerosis, or tissue ischemia (no data). Thus, 6H-thieno[2,3-b]pyrrole-5-carboxylic acid and (3S)-amino-1-((3R,4S)-dihydroxypyrrolidin-1-yl)-(2R)-hydroxy-4-phenylbutan-1-one were coupled using 4-(dimethylamino)pyridine, 1-hydroxybenzotriazole hydrate and 1-(3-dimethylaminopropyl)-3-ethylcarbodiimide hydrochloride in CH2Cl2/DMF to give 6H-thieno[2,3-b]pyrrole-5-carboxylic acid [(1S)-benzyl-3-((3R,4S)-dihydroxypyrrolidin-1-yl)-(2R)-hydroxy-3-oxopropyl]amide.

ST pyrrolylcarboxamide fused prepn glycogen phosphorylase inhibitor; thienopyrrolecarboxamide prepn antidiabetic; diabetes insulin resistance diabetic neuropathy treatment fused pyrrolecarboxamide; diabetic nephropathy retinopathy cataract hyperglycemia hypercholesterolemia hypertension treatment pyrrolecarboxamide; hyperinsulinemia hyperlipidemia atherosclerosis tissue ischemia treatment fused pyrrolecarboxamide

IT Ischemia
(cardiac, treatment; preparation of fused pyrrolylcarboxamides as glycogen phosphorylase inhibitors)

IT Antiobesity agents
a2-Adrenoceptor antagonists
b-Adrenoceptor agonists
(coadministration; preparation of fused pyrrolylcarboxamides as glycogen phosphorylase inhibitors)

IT Sulfonylureas
RL: THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(coadministration; preparation of fused pyrrolylcarboxamides as glycogen phosphorylase inhibitors)

IT Kidney, disease
(diabetic nephropathy, treatment; preparation of fused pyrrolylcarboxamides as glycogen phosphorylase inhibitors)

IT Nerve, disease
(diabetic neuropathy, treatment; preparation of fused pyrrolylcarboxamides as glycogen phosphorylase inhibitors)

IT Eye, disease
(diabetic retinopathy, treatment; preparation of fused pyrrolylcarboxamides as glycogen phosphorylase inhibitors)

IT Antioxidants
(fatty acid oxidation inhibitors coadministration; preparation of fused pyrrolylcarboxamides as glycogen phosphorylase inhibitors)

IT Gluconeogenesis
(inhibitors coadministration; preparation of fused pyrrolylcarboxamides as glycogen phosphorylase inhibitors)

IT Heart, disease
(ischemia, treatment; preparation of fused pyrrolylcarboxamides as glycogen phosphorylase inhibitors)

IT Anti-ischemic agents
Anticholesteremic agents
Antidiabetic agents
Antihypertensives
Drug delivery systems
Human
Hypolipemic agents
(preparation of fused pyrrolylcarboxamides as glycogen phosphorylase inhibitors)

IT Atherosclerosis
Cataract
Diabetes mellitus
Hypercholesterolemia
Hyperglycemia
Hypertension
Hypertriglyceridemia
Ischemia
(treatment; preparation of fused pyrrolylcarboxamides as glycogen phosphorylase inhibitors)

IT Hyperlipidemia
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(treatment; preparation of fused pyrrolylcarboxamides as glycogen phosphorylase inhibitors)

IT Peroxisome proliferator-activated receptors
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(g, PPAR-g agonists coadministration; preparation of fused pyrrolylcarboxamides as glycogen phosphorylase inhibitors)

IT 9007-92-5, Glucagon, biological studies 106602-62-4, Amylin
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(antagonists coadministration; preparation of fused pyrrolylcarboxamides as glycogen phosphorylase inhibitors)

IT 56-03-1D, Biguanide, derivs. 64-77-7, Tolbutamide 94-20-2, Chloropropamide 114-86-3, Phenformin 458-24-2, Fenfluramine 657-24-9, Metformin 692-13-7, Buformin 968-81-0, Acetohexamide 1156-19-0, Tolazamide 2295-31-0D, Thiazolidinedione, derivs. 7440-62-2D, Vanadium, complexes 9004-10-8, Insulin, biological studies 9004-10-8D, Insulin, analogs 10238-21-8, Glibenclamide 12179-36-1D, Pervanadyl (VO(O2)+), complexes 23602-78-0, Benfluorex 28299-33-4D, Imidazoline, derivs. 29094-61-9, Glipizide 37353-31-4, Vanadate 51037-30-0, Acipimox 51110-01-1D, Somatostatin, analogs 54870-28-9, Meglitinide 56180-94-0, Acarbose 66529-17-7, Midaglizole 72432-03-2, Miglitol 74772-77-3, Ciglitazone 75358-37-1, Linogliride 79944-58-4, Idazoxan 80879-63-6, Emiglitate 83480-29-9, Voglibose 86615-96-5, BRL35135 88431-47-4, Clomoxir 89197-32-0, Efaroxan 90505-66-1, Ro 16-8714 90730-96-4, BRL37344 93479-97-1, Glimepiride 97322-87-7, Troglitazone 104343-33-1, MDL-25637 105182-45-4, Fluparoxan 105816-04-4, Nateglinide 106612-94-6, Human GLP-1 -(7-37) 107444-51-9, Rat GLP-1(7-36)amide 109229-58-5, Englitazone 110605-64-6, Isaglidole 111025-46-8, Pioglitazone 115656-32-1, D 7114 122320-73-4, Rosiglitazone 122575-28-4, Naglivan 122830-14-2, Deriglidole 124083-20-1, Etomoxir 127214-23-7, Camiglibose 130714-47-5, WAG 994 133107-64-9, Insulin lispro 135062-02-1, Repaglinide 138908-40-4, CL 316243 141200-24-0, Darglitazone 141758-74-9, AC2993 187887-46-3, Symlin 395214-16-1
RL: THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(coadministration; preparation of fused pyrrolylcarboxamides as glycogen phosphorylase inhibitors)

IT 9001-42-7, a-Glucosidase 9025-82-5, Phosphodiesterase
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(inhibitors coadministration; preparation of fused pyrrolylcarboxamides as glycogen phosphorylase inhibitors)

IT 9035-74-9, Glycogen phosphorylase
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(inhibitors; preparation of fused pyrrolylcarboxamides as glycogen phosphorylase inhibitors)

IT 332098-11-0P 332098-12-1P 332098-13-2P 332098-14-3P 332098-15-4P 332098-16-5P 332098-17-6P 332098-18-7P 332098-19-8P 332098-20-1P 332098-21-2P 332098-22-3P 332098-23-4P 332098-24-5P 332098-25-6P 332098-26-7P 332098-27-8P 332098-28-9P 332098-29-0P 332098-30-3P 332098-31-4P 332098-32-5P 332098-33-6P 332098-34-7P 332098-35-8P 332098-36-9P 332098-37-0P 332098-38-1P 332098-39-2P 332098-40-5P 332098-41-6P 332098-42-7P 332098-43-8P 332098-44-9P 332098-45-0P 332098-46-1P 332098-47-2P 332098-48-3P 332098-49-4P 332098-50-7P

332098-52-9P 332098-54-1P 332098-55-2P 332098-57-4P 332098-59-6P
332098-61-0P 332098-63-2P 332098-65-4P
RL: PAC (Pharmacological activity); SPN (Synthetic preparation); THU (Therapeutic use); BIOL (Biological study); PREP (Preparation); USES (Uses)
(preparation of fused pyrrolylcarboxamides as glycogen phosphorylase inhibitors)

IT 637-81-0, Azidoacetic acid ethyl ester 1066-54-2, (Trimethylsilyl)acetylene 1126-09-6, Piperidine-4-carboxylic acid ethyl ester 4530-18-1, Boc-DL-Phenylalanine 6030-36-0, 4-Methylthiophene-2-carboxaldehyde 7283-96-7, 5-Chlorothiophene-2-carboxaldehyde 13679-70-4, 5-Methyl-2-thiophenecarboxaldehyde 13734-34-4, Boc-L-Phenylalanine 14345-97-2, 2-Chloro-3-methylthiophene 17186-57-1 18791-75-8, 4-Bromothiophene-2-carboxaldehyde 21508-19-0, 5-Chlorofuran-2-carboxaldehyde 21921-76-6, 4-Bromo-2-furaldehyde 24445-35-0 29669-49-6, 5-Fluorothiophene-2-carboxaldehyde 31486-85-8, Thieno[2,3-b]thiophene-2-carboxaldehyde 35357-56-3, 6H-Thieno[2,3-b]pyrrole-5-carboxylic acid ethyl ester 39793-31-2, 4H-Thieno[3,2-b]pyrrole-5-carboxylic acid 51856-25-8, 6H-Thieno[2,3-b]pyrrole-5-carboxylic acid 51856-29-2, 2-Formyl-6H-thieno[2,3-b]pyrrole-5-carboxylic acid ethyl ester 57500-51-3, 4-Chlorothiophene-2-carboxaldehyde 58963-45-4, 2-Formyl-6H-thieno[2,3-b]pyrrole-5-carboxylic acid 59958-27-9, 2-Formyl-4H-thieno[3,2-b]pyrrole-5-carboxylic acid ethyl ester 62023-60-3, (2R,3S)-3-Benzyloxycarbonylamino-2-hydroxy-4-phenylbutyric acid 80709-80-4, 2-Methyl-4H-furo[3,2-b]pyrrole-5-carboxylic acid 80709-83-7, 2-Bromo-4H-furo[3,2-b]pyrrole-5-carboxylic acid ethyl ester 91545-55-0, 2-Bromo-6H-thieno[2,3-b]pyrrole-5-carboxylic acid ethyl ester 105181-72-4, (2R,3S)-3-tert-Butoxycarbonylamino-2-hydroxy-4-phenylbutyric acid 153548-49-3 164667-45-2, 2-Formyl-4H-furo[3,2-b]pyrrole-5-carboxylic acid 186431-46-9 186432-05-3 238749-50-3, 2-Bromo-4H-thieno[3,2-b]pyrrole-5-carboxylic acid ethyl ester 519188-80-8
RL: RCT (Reactant); RACT (Reactant or reagent)
(preparation of fused pyrrolylcarboxamides as glycogen phosphorylase inhibitors)

IT 65782-04-9P, 5-Chloro-4-methylthiophene-2-carboxaldehyde 238749-54-7P, 2-Methyl-4H-thieno[3,2-b]pyrrole-5-carboxylic acid ethyl ester 332098-79-0P 332098-81-4P 332098-83-6P, 2-Bromo-6H-thieno[2,3-b]pyrrole-5-carboxylic acid 332098-85-8P, 2-Methyl-6H-thieno[2,3-b]pyrrole-5-carboxylic acid ethyl ester 332098-87-0P, 2-Methyl-6H-thieno[2,3-b]pyrrole-5-carboxylic acid 332098-89-2P 332098-91-6P 332098-93-8P 332098-95-0P 332098-97-2P 332098-99-4P 332099-01-1P, 2-Chloro-6H-thieno[2,3-b]pyrrole-5-carboxylic acid ethyl ester 332099-03-3P, 2-Chloro-6H-thieno[2,3-b]pyrrole-5-carboxylic acid 332099-05-5P, 2,4-Dichloro-6H-thieno[2,3-b]pyrrole-5-carboxylic acid ethyl ester 332099-07-7P, 2,4-Dichloro-6H-thieno[2,3-b]pyrrole-5-carboxylic acid 332099-09-9P, 2-Bromo-4H-thieno[3,2-b]pyrrole-5-carboxylic acid 332099-11-3P, 2-Bromo-4H-furo[3,2-b]pyrrole-5-carboxylic acid 332099-14-6P, 2-Methyl-4H-thieno[3,2-b]pyrrole-5-carboxylic acid 332099-16-8P, 2-Cyano-6H-thieno[2,3-b]pyrrole-5-carboxylic acid 332099-18-0P 332099-20-4P 332099-22-6P, 2-Fluoro-4H-thieno[3,2-b]pyrrole-5-carboxylic acid ethyl ester 332099-24-8P, 2-Fluoro-4H-thieno[3,2-b]pyrrole-5-carboxylic acid 332099-26-0P, 2-Cyano-4H-furo[3,2-b]pyrrole-5-carboxylic acid 332099-28-2P, 2-Chloro-4H-furo[3,2-b]pyrrole-5-carboxylic acid ethyl ester 332099-29-3P, 2-Chloro-4H-furo[3,2-b]pyrrole-5-carboxylic acid 332099-31-7P, 3-Chloro-4H-thieno[3,2-b]pyrrole-5-carboxylic acid ethyl ester 332099-33-9P, 3-Chloro-4H-thieno[3,2-b]pyrrole-5-carboxylic acid 332099-35-1P, 3-Bromo-4H-thieno[3,2-b]pyrrole-5-carboxylic acid ethyl ester 332099-36-2P, 3-Bromo-4H-thieno[3,2-b]pyrrole-5-carboxylic acid 332099-38-4P, 2-Chloro-4H-thieno[3,2-b]pyrrole-5-carboxylic acid ethyl ester 332099-40-8P, 2-Chloro-4H-thieno[3,2-b]pyrrole-5-carboxylic acid 332099-42-0P, 3-Methyl-4H-thieno[3,2-b]pyrrole-5-carboxylic acid ethyl ester 332099-44-2P, 3-Methyl-4H-thieno[3,2-b]pyrrole-5-carboxylic acid 332099-46-4P, 2-Formyl-4H-thieno[3,2-b]pyrrole-5-carboxylic acid 332099-48-6P, 2-Cyano-4H-thieno[3,2-b]pyrrole-5-carboxylic acid 332099-50-0P, 3-Bromo-4H-furo[3,2-b]pyrrole-5-carboxylic acid ethyl ester 332099-52-2P, 3-Bromo-4H-furo[3,2-b]pyrrole-5-carboxylic acid 332099-54-4P 332099-55-5P 332099-56-6P, 2-Chloro-3-methyl-4H-thieno[3,2-b]pyrrole-5-carboxylic acid ethyl ester 332099-58-8P, 2-Chloro-3-methyl-4H-thieno[3,2-b]pyrrole-5-carboxylic acid 332099-60-2P 332099-62-4P
RL: RCT (Reactant); SPN (Synthetic preparation); PREP (Preparation); RACT (Reactant or reagent)
(preparation of fused pyrrolylcarboxamides as glycogen phosphorylase inhibitors)

RE.CNT 3 THERE ARE 3 CITED REFERENCES AVAILABLE FOR THIS RECORD

RE

(1) Esteve, L; ES 2081747 A 1996 CAPLUS
(2) Hitzel, V; US 4325963 A 1982 CAPLUS
(3) Pfizer; EP 0846464 A 1998 CAPLUS

L33 ANSWER 3 OF 9 CAPLUS COPYRIGHT 2007 ACS on STN
ACCESSION NUMBER: 2003:570833 CAPLUS <<LOGINID::20070124>>
DOCUMENT NUMBER: 139:111682
TITLE: Combined use of a GLP-1 compound and a modulator of diabetic late complications
INVENTOR(S): Knudsen, Lotte Bjerre; Selmer, Johan
PATENT ASSIGNEE(S): Novo Nordisk A/S, Den.
SOURCE: PCT Int. Appl., 22 pp.
CODEN: PIXXD2

DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2003059372	A2	20030724	WO 2002-DK888	20021220 <--
WO 2003059372	A3	20040325		
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, UZ, VC, VN, YU, ZA, ZM, ZW				
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US 2003144206	A1	20030731	US 2002-328282	20021223 <--

PRIORITY APPLN. INFO.: DK 2001-1969 A 20011229
DK 2002-760 A 20020517
DK 2001-969 A 20011229
US 2002-350087P P 20020117
WO 2002-DK888 W 20021220

AN 2003:570833 CAPLUS <<LOGINID::20070124>>
DN 139:111682
ED Entered STN: 25 Jul 2003
TI Combined use of a GLP-1 compound and a modulator of diabetic late complications
IN Knudsen, Lotte Bjerre; Selmer, Johan
PA Novo Nordisk A/S, Den.
SO PCT Int. Appl., 22 pp.
CODEN: PIXXD2
DT Patent
LA English
IC ICM A61K038-00
CC 1-10 (Pharmacology)
Section cross-reference(s): 2, 63
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI WO 2003059372	A2	20030724	WO 2002-DK888	20021220 <--
WO 2003059372	A3	20040325		
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PRAI DK 2001-1969 A 20011229
DK 2002-760 A 20020517
DK 2001-969 A 20011229
US 2002-350087P P 20020117
WO 2002-DK888 W 20021220

CLASS

PATENT NO.	CLASS	PATENT FAMILY CLASSIFICATION CODES
WO 2003059372	ICM	A61K038-00
	IPCI	A61K0038-00 [ICM,7]
	IPCR	A61K0045-00 [LC*]; A61K0045-00 [LA]; A61K0031-138 [LC*]; A61K0031-138 [LA]; A61K0031-165 [LC*]; A61K0031-165 [LA]; A61K0031-167 [LC*]; A61K0031-167 [LA]; A61K0031-21 [LC*]; A61K0031-216 [LA]; A61K0031-35 [LC*]; A61K0031-35 [LA]; A61K0031-401 [LC*]; A61K0031-401 [LA]; A61K0031-403 [LC*]; A61K0031-403 [LA]; A61K0031-404 [LA]; A61K0031-407 [LC*]; A61K0031-407 [LA]; A61K0031-4164 [LC*]; A61K0031-4166 [LA]; A61K0031-4184 [LA]; A61K0031-4188 [LA]; A61K0031-4196 [LC*]; A61K0031-4196 [LA]; A61K0031-472 [LC*]; A61K0031-472 [LA]; A61K0031-5375 [LC*]; A61K0031-5377 [LA]; A61K0031-55 [LC*]; A61K0031-55 [LA]; A61K0038-00 [LC*]; A61K0038-00 [LA]; A61K0038-26 [LC*]; A61K0038-26 [LA]; A61P0003-00 [LC*]; A61P0003-10 [LA]; A61P0009-00 [LC*]; A61P0009-10 [LA]; A61P0009-12 [LA]; A61P0013-00 [LC*]; A61P0013-12 [LA]; A61P0025-00

[I,C*]; A61P0025-00 [I,A]; A61P0025-02 [I,A];
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AU 2002351753 IPCI A61K0038-00 [ICM,7]
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EP 1461070 IPCI A61K0038-26 [ICM,7]; A61K0031-35 [ICS,7]; A61P0003-10
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FTERM 4C084/AA02; 4C084/AA03; 4C084/AA19; 4C084/BA19;
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4C206/ZC20; 4C206/ZC35; 4C206/ZC42

US 2003144206 IPCI A61K0038-26 [ICM,7]; A61K0031-401 [ICS,7]
IPCR A61K0031-401 [I,C*]; A61K0031-401 [I,A]; A61K0038-26
[I,C*]; A61K0038-26 [I,A]
NCL 514/012.000; 514/423.000

AB Methods and uses for treatment of diabetic late complications comprising administration of a GLP-1 compound and a modulator of diabetic complications.

ST GLP1 diabetes late complication therapy; glucagon like peptide I analog fragment antidiabetic

IT Angiotensin receptor antagonists
Antihypertensives
Human
Hypertension
Protein sequences
b-Adrenoceptor antagonists
b1-Adrenoceptor antagonists
(combined use of a GLP-1 compound and a modulator of diabetic late complications)

IT Kidney, disease
(diabetic nephropathy; combined use of a GLP-1 compound and a modulator of diabetic late complications)

IT Nerve, disease
(diabetic neuropathy; combined use of a GLP-1 compound and a modulator of diabetic late complications)

IT Eye, disease
(diabetic retinopathy; combined use of a GLP-1 compound and a modulator of diabetic late complications)

IT Gene, animal
RL: PAC (Pharmacological activity); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(glp-1; combined use of a GLP-1 compound and a modulator of diabetic late complications)

IT Diabetes mellitus
(non-insulin-dependent; combined use of a GLP-1 compound and a modulator of diabetic late complications)

IT Antidiabetic agents
Drug delivery systems
(oral; combined use of a GLP-1 compound and a modulator of diabetic late complications)

IT Drug delivery systems
(parenterals; combined use of a GLP-1 compound and a modulator of diabetic late complications)

IT 496765-91-4
RL: PAC (Pharmacological activity); PRP (Properties); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(amino acid sequence; combined use of a GLP-1 compound and a modulator of diabetic late complications)

IT 525-66-6, Propranolol 13523-86-9, Pindolol 26839-75-8, Timolol 29122-68-7, Atenolol 37517-30-9, Acebutolol 42200-33-9, Nadolol 51384-51-1, Metoprolol 62571-86-2, Captopril 75847-73-3, Enalapril 76547-98-3, Lisinopril 81147-92-4, Esmolol 83647-97-6, Spirapril 85441-61-8, Quinapril 86541-75-5, Benazepril 87333-19-5, Ramipril 87679-37-6, Trandolapril 89371-37-9, Irnidapril 89750-14-1D, GLP-1, analogs or fragments 98048-97-6, Fosinopril 107133-36-8, Perindopril erbumine 114798-26-4, Losartan 135038-57-2, Alatriopril 136087-85-9, Fidarestat 137862-53-4, Valsartan 138402-11-6, Irbesartan 141732-76-5, Exendin-4 141732-76-5D, Exendin-4, derivs. 169939-94-0, Ly 333531
RL: PAC (Pharmacological activity); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(combined use of a GLP-1 compound and a modulator of diabetic late complications)

IT 9015-82-1, Angiotensin-converting enzyme 9028-31-3, Aldose reductase 141436-78-4, Protein kinase C
RL: PAC (Pharmacological activity); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(inhibitors; combined use of a GLP-1 compound and a modulator of diabetic late complications)

L33 ANSWER 4 OF 9 CAPLUS COPYRIGHT 2007 ACS on STN
ACCESSION NUMBER: 2003:320036 CAPLUS <<LOGINID::20070124>>
DOCUMENT NUMBER: 138:338498
TITLE: Preparation of human glucagon-like-peptide-1 mimics and their use in the treatment of diabetes and related conditions
INVENTOR(S): Natarajan, Sesha I.; Bastos, Margarita M.; Bernatowicz, Michael S.; Mapelli, Claudio; Lee, Ving; Ewing, William R.
PATENT ASSIGNEE(S): Bristol-Myers Squibb Company, USA
SOURCE: PCT Int. Appl., 153 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English

FAMILY ACC. NUM. COUNT: 2
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2003033671	A2	20030424	WO 2002-US33386	20021018 <--
WO 2003033671	A3	20051229		

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CA 2463908	A1	20030424	CA 2002-2463908	20021018 <--
JP 2005514337	T	20050519	JP 2003-536401	20021018
CN 1630709	A	20050622	CN 2002-820558	20021018
EP 1572892	A2	20050914	EP 2002-782185	20021018

R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR, BG, CZ, EE, SK

BR 2002013377	A	20060523	BR 2002-13377	20021018
NO 2004001203	A	20040610	NO 2004-1203	20040323
ZA 2004002846	A	20050816	ZA 2004-2846	20040415
PRIORITY APPLN. INFO.:			US 2001-342015P	P 20011018
			WO 2002-US33386	W 20021018

OTHER SOURCE(S): MARPAT 138:338498
AN 2003:320036 CAPLUS <<LOGINID::20070124>>
DN 138:338498
ED Entered STN: 25 Apr 2003
TI Preparation of human glucagon-like-peptide-1 mimics and their use in the treatment of diabetes and related conditions
IN Natarajan, Sesha I.; Bastos, Margarita M.; Bernatowicz, Michael S.; Mapelli, Claudio; Lee, Ving; Ewing, William R.
PA Bristol-Myers Squibb Company, USA
SO PCT Int. Appl., 153 pp.
CODEN: PIXXD2
DT Patent
LA English
IC ICM C12N
CC 34-3 (Amino Acids, Peptides, and Proteins)
Section cross-reference(s): 1, 63
FAN.CNT 2

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI WO 2003033671	A2	20030424	WO 2002-US33386	20021018 <--
WO 2003033671	A3	20051229		

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RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG

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JP 2005514337	T	20050519	JP 2003-536401	20021018
CN 1630709	A	20050622	CN 2002-820558	20021018
EP 1572892	A2	20050914	EP 2002-782185	20021018

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BR 2002013377	A	20060523	BR 2002-13377	20021018
NO 2004001203	A	20040610	NO 2004-1203	20040323
ZA 2004002846	A	20050816	ZA 2004-2846	20040415
PRAI US 2001-342015P	P	20011018		
WO 2002-US33386	W	20021018		

CLASS

PATENT NO.	CLASS	PATENT FAMILY CLASSIFICATION CODES
WO 2003033671	ICM	C12N
	IPCI	C12N [ICM,7]
	IPCR	A61K0038-00 [I,C*]; A61K0038-00 [I,A]; A61K0038-26 [I,C*]; A61K0038-26 [I,A]; A61P0003-00 [I,C*]; A61P0003-04 [I,A]; A61P0003-06 [I,A]; A61P0003-10 [I,A]; A61P0005-00 [I,C*]; A61P0005-50 [I,A]; A61P0009-00 [I,C*]; A61P0009-10 [I,A]; A61P0009-12 [I,A]; A61P0013-00 [I,C*]; A61P0013-12 [I,A]; A61P0017-00 [I,C*]; A61P0017-02 [I,A]; A61P0025-00 [I,C*]; A61P0025-00 [I,A]; A61P0027-00 [I,C*]; A61P0027-02 [I,A]; A61P0043-00 [I,C*]; A61P0043-00 [I,A]; C07K0007-00 [I,C*]; C07K0007-06 [I,A]; C07K0007-08 [I,A]; C07K0014-00 [I,C*]; C07K0014-00 [I,A]; C07K0014-435 [I,C*]; C07K0014-605 [I,A]
	ECLA	C07K014/605
CA 2463908	IPCI	C07K0014-605 [ICM,7]; C07K0014-435 [ICM,7,C*]; C07K0007-06 [ICS,7]; C07K0007-00 [ICS,7,C*]; A61K0038-08 [ICS,7]; A61K0038-26 [ICS,7]
	IPCR	A61K0038-00 [I,C*]; A61K0038-00 [I,A]; A61K0038-26 [I,C*]; A61K0038-26 [I,A]; A61P0003-00 [I,C*]; A61P0003-04 [I,A]; A61P0003-06 [I,A]; A61P0003-10 [I,A]; A61P0005-00 [I,C*]; A61P0005-50 [I,A]; A61P0009-00 [I,C*]; A61P0009-10 [I,A]; A61P0009-12 [I,A]; A61P0013-00 [I,C*]; A61P0013-12 [I,A]; A61P0017-00 [I,C*]; A61P0017-02 [I,A]; A61P0025-00 [I,C*]; A61P0025-00 [I,A]; A61P0027-00 [I,C*]; A61P0027-02 [I,A]; A61P0043-00 [I,C*]; A61P0043-00 [I,A]; C07K0007-00 [I,C*]; C07K0007-06 [I,A]; C07K0007-08 [I,A]; C07K0014-00 [I,C*]; C07K0014-00 [I,A]; C07K0014-435 [I,C*]; C07K0014-605 [I,A]
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	IPCR	A61K0038-00 [N,A]; A61K0038-00 [N,C*]; C07K0014-435 [I,C*]; C07K0014-605 [I,A]
	FTERM	4C084/AA02; 4C084/AA06; 4C084/AA07; 4C084/BA01; 4C084/BA08; 4C084/BA17; 4C084/BA23; 4C084/BA32; 4C084/CA59; 4C084/DB35; 4C084/MA01; 4C084/NA14; 4C084/ZA012; 4C084/ZA332; 4C084/ZA452; 4C084/ZA702; 4C084/ZA812; 4C084/ZA892; 4C084/ZC032; 4C084/ZC332; 4C084/ZC352; 4C084/ZC412; 4H045/AA10; 4H045/AA30; 4H045/BA10; 4H045/BA16; 4H045/BA17; 4H045/BA18; 4H045/DA37; 4H045/EA20; 4H045/FA10; 4H045/FA20; 4H045/FA33; 4H045/FA34; 4H045/GA21
CN 1630709	IPCI	C12N0001-00 [ICM,7]
	IPCR	A61K0038-00 [I,C*]; A61K0038-00 [I,A]; A61K0038-26 [I,C*]; A61K0038-26 [I,A]; A61P0003-00 [I,C*]; A61P0003-04 [I,A]; A61P0003-06 [I,A]; A61P0003-10 [I,A]; A61P0005-00 [I,C*]; A61P0005-50 [I,A]; A61P0009-00 [I,C*]; A61P0009-10 [I,A]; A61P0009-12 [I,A]; A61P0013-00 [I,C*]; A61P0013-12 [I,A]; A61P0017-00 [I,C*]; A61P0017-02 [I,A]; A61P0025-00 [I,C*]; A61P0025-00 [I,A]; A61P0027-00 [I,C*]; A61P0027-02 [I,A]; A61P0043-00 [I,C*]; A61P0043-00 [I,A]; C07K0007-00 [I,C*]; C07K0007-06 [I,A]; C07K0007-08 [I,A]; C07K0014-00 [I,C*]; C07K0014-00 [I,A]; C07K0014-435 [I,C*]; C07K0014-605 [I,A]
	ECLA	C07K014/605
EP 1572892	IPCI	C12N0001-00 [ICM,7]
	IPCR	A61K0038-00 [I,C*]; A61K0038-00 [I,A]; A61K0038-08 [I,C]; A61K0038-08 [I,A]; A61K0038-26 [I,C*]; A61K0038-26 [I,A]; A61P0003-00 [I,C*]; A61P0003-04 [I,A]; A61P0003-06 [I,A]; A61P0003-10 [I,A]; A61P0005-00 [I,C*]; A61P0005-50 [I,A]; A61P0009-00 [I,C*]; A61P0009-10 [I,A]; A61P0009-12 [I,A]; A61P0013-00 [I,C*]; A61P0013-12 [I,A]; A61P0017-00 [I,C*]; A61P0017-02 [I,A]; A61P0025-00 [I,C*]; A61P0025-00 [I,A]; A61P0027-00 [I,C*]; A61P0027-02 [I,A]; A61P0043-00 [I,C*]; A61P0043-00 [I,A]; C07K0002-00 [I,C]; C07K0002-00 [I,A]; C07K0007-00 [I,C*]; C07K0007-06 [I,A]; C07K0007-08 [I,A]; C07K0014-00 [I,C*]; C07K0014-00 [I,A]; C07K0014-435 [I,C*]; C07K0014-605 [I,A]
	ECLA	C07K014/605
BR 2002013377	IPCI	A61K0038-00 [ICS,7]; A61K0038-08 [ICS,7]; C07K0002-00 [ICS,7]
	IPCR	A61K0038-00 [N,C*]; C07K0014-435 [I,C*]; A61K0038-00 [N,A]; C07K0014-605 [I,A]
	ECLA	C07K014/605
NO 2004001203	IPCI	C07K0014-605 [ICM,7]; C07K0014-435 [ICM,7,C*]; C07K0004-00 [ICS,7]; A61K0038-26 [ICS,7]; A61K0038-08 [ICS,7]; A61K0038-10 [ICS,7]
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	ECLA	C07K014/605
ZA 2004002846	IPCR	A61K0038-00 [N,C*]; C07K0014-435 [I,C*]; A61K0038-00 [N,A]; C07K0014-605 [I,A]
	ECLA	C07K014/605

OS MARPAT 138:338498
AB The invention provides novel human glucagon-like peptide-1 (GLP-1) peptide mimics A-Xaa1-Xaa2-Xaa3-Xaa4-Xaa5-Xaa6-Xaa7-Xaa8-Xaa9-Y-Z-B [Xaa1-Xaa9 are naturally or non-naturally occurring amino acid residues; Y and Z are amino acid residues which may be substituted; A and B are optionally present; A is H, an amino acid or peptide containing .apprx.

1-15 amino acid residues, an R group [H, (cyclo)alkyl, cycloalkylalkyl, heterocyclyl, heterocycloalkyl, (hetero)aryl, arylalkyl, aryloxyalkyl, heteroarylalkyl, or heteroaryloxyalkyl], an RCO (amide) group, a carbamate group, a urea, a sulfonamido, or an aminosulfonyl group; B is OH, alkoxy, etc., an amino or amino acid residue, or a peptide containing from 1-15 amino acid residues, terminating at the C-terminus as a carboxamide, ester, carboxyl, or an amino alc.] that mimic the biol. activity of the native GLP-1 peptide and thus are useful for the treatment or prevention of diseases or disorders associated with GLP activity. These chemical-modified peptides stimulate insulin secretion in type II diabetics and produce other beneficial insulinotropic responses, while exhibiting increased stability to proteolytic cleavage making them ideal therapeutic candidates for oral or parenteral administration. A method of preparing the polypeptides comprises replacing the message sequence of the polypeptide with a variant message sequence capable of inducing receptor mediated signal transduction. An example is claimed peptide H-AEGTFTSD-Bip(2-Et)-Bip(2-Me)-NH2 (Bip = biphenylalanine residue).

ST glucagon like peptide mimic prepn treatment diabetes

IT Antiarteriosclerotics
(antiatherosclerotics; preparation of human glucagon-like-peptide-1 mimics for use in treatment of diabetes and related conditions)

IT Kidney, disease
(diabetic nephropathy; preparation of human glucagon-like-peptide-1 mimics for use in treatment of diabetes and related conditions)

IT Nerve, disease
(diabetic neuropathy; preparation of human glucagon-like-peptide-1 mimics for use in treatment of diabetes and related conditions)

IT Eye, disease
(diabetic retinopathy; preparation of human glucagon-like-peptide-1 mimics for use in treatment of diabetes and related conditions)

IT Metabolic disorders
(metabolic syndrome X; preparation of human glucagon-like-peptide-1 mimics for use in treatment of diabetes and related conditions)

IT Antidiabetic agents
Antihypertensives
Antiobesity agents
Atherosclerosis
Diabetes mellitus
Human
Hyperglycemia
Hypertension
Hypertriglyceridemia
Hypolipemic agents
Obesity
Wound healing
(preparation of human glucagon-like-peptide-1 mimics for use in treatment of diabetes and related conditions)

IT Hyperlipidemia
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(preparation of human glucagon-like-peptide-1 mimics for use in treatment of diabetes and related conditions)

IT Peptides, preparation
RL: PAC (Pharmacological activity); SPN (Synthetic preparation); THU (Therapeutic use); BIOL (Biological study); PREP (Preparation); USES (Uses)
(preparation of human glucagon-like-peptide-1 mimics for use in treatment of diabetes and related conditions)

IT 89750-14-1DP, Glucagon-like peptide I, mimics 516514-32-2P
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516521-55-4P
RL: PAC (Pharmacological activity); SPN (Synthetic preparation); THU (Therapeutic use); BIOL (Biological study); PREP (Preparation); USES (Uses)
(preparation of human glucagon-like-peptide-1 mimics for use in treatment of diabetes and related conditions)

IT 150-46-9, Triethyl borate 1973-22-4, 1 Bromo 2 ethylbenzene 4326-36-7 16419-60-6, o Tolylboronic acid 93267-04-0 516521-49-6
RL: RCT (Reactant); RACT (Reactant or reagent)
(preparation of human glucagon-like-peptide-1 mimics for use in treatment of diabetes and related conditions)

IT 90002-36-1P, 2 Ethylphenylboronic acid 112766-18-4P 516521-46-3P 516521-47-4P 516521-48-5P 516521-50-9P 516521-51-0P
RL: RCT (Reactant); SPN (Synthetic preparation); PREP (Preparation); RACT (Reactant or reagent)
(preparation of human glucagon-like-peptide-1 mimics for use in treatment of diabetes and related conditions)

IT 51-64-9, Dexamphetamine 94-20-2, Chloropropamide 122-09-8, Phentermine 637-07-0, Clofibrate 657-24-9, Metformin 9004-10-8, Insulin, biological studies 10238-21-8, Glyburide 14838-15-4, Phenylpropanolamine 21187-98-4, Gliclazide 22232-71-9, Mazindol 25812-30-0, Gemfibrozil 29094-61-9, Glipizide 49562-28-9, Fenofibrate 56180-94-0, Acarbose 72432-03-2, Miglitol 75330-75-5, Lovastatin 79902-63-9, Simvastatin 81093-37-0, Pravastatin 93479-97-1, Glimepiride 93957-54-1, Fluvastatin 96829-58-2, Orlistat 97240-79-4, Topiramate 97322-87-7, Troglitazone 105816-04-4, Nateglinide 106650-56-0, Sibutramine 111025-46-8, Pioglitazone 122320-73-4, Rosiglitazone 134523-00-5, Atorvastatin 135062-02-1, Repaglinide 141750-63-2, Nisvastatin 141758-74-9, AC 2993 144288-97-1, TS-962 145599-86-6, Cerivastatin 152755-31-2, LY295427 159183-92-3, L750355 161600-01-7, Isaglitazone 166518-60-1, Avasimibe 170861-63-9, JTT-501 176435-10-2, LY315902 178759-95-0, MD 700 196808-45-4 199113-98-9 199914-96-0, YM-440 213252-19-8, KRP297 244081-42-3, AJ9677 258345-41-4, GW-409544 262352-17-0, CP 529414 282526-98-1, ATL-962 287714-41-4 335149-08-1, L895645 335149-14-9, R-119702 335149-15-0, KAD1129 335149-17-2, ARHO39242 335149-23-0, NVPDPP-728A 335149-25-2,
CP331648 430433-17-3, Glipyride 444069-80-1, Axokine
RL: THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(preparation of human glucagon-like-peptide-1 mimics for use in treatment of diabetes and related conditions)

L33 ANSWER 5 OF 9 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on STN
ACCESSION NUMBER: 2003:390202 BIOSIS <<LOGINID::20070124>>
DOCUMENT NUMBER: PREV200300390202
TITLE: The glucagon-like peptides: A double-edged therapeutic sword?.
AUTHOR(S): Perry, TracyAnn [Reprint Author]; Greig, Nigel H.
CORPORATE SOURCE: Section of Drug Design and Development, Laboratory of Neurosciences, Gerontology Research Center, National Institute on Aging, National Institutes of Health, 5600 Nathan Shock Drive, Baltimore, MD, 21224, USA
perryt@grc.nia.nih.gov
SOURCE: Trends in Pharmacological Sciences, (July 2003) Vol. 24, No. 7, pp. 377-383. print.
ISSN: 0165-6147 (ISSN print).
DOCUMENT TYPE: Article
General Review; (Literature Review)
LANGUAGE: English
ENTRY DATE: Entered STN: 27 Aug 2003
Last Updated on STN: 27 Aug 2003
AN 2003:390202 BIOSIS <<LOGINID::20070124>>
DN PREV200300390202
TI The glucagon-like peptides: A double-edged therapeutic sword?.
AU Perry, TracyAnn [Reprint Author]; Greig, Nigel H.
CS Section of Drug Design and Development, Laboratory of Neurosciences, Gerontology Research Center, National Institute on Aging, National Institutes of Health, 5600 Nathan Shock Drive, Baltimore, MD, 21224, USA
perryt@grc.nia.nih.gov
SO Trends in Pharmacological Sciences, (July 2003) Vol. 24, No. 7, pp. 377-383. print.
ISSN: 0165-6147 (ISSN print).
DT Article
General Review; (Literature Review)
LA English
ED Entered STN: 27 Aug 2003
Last Updated on STN: 27 Aug 2003
AB Glucagon-like peptide-1(7-36)-amide (GLP-1) is an endogenous peptide that is secreted from the gut in response to the presence of food. Recent studies have established that GLP-

1 and its longer-acting analog exendin-4 have multiple synergistic effects on glucose-dependent, insulin secretion pathways of the pancreatic beta-cell and on plasticity in neuronal cells. Recent interest has focused on the development of these peptides as a novel therapeutic strategy for non-insulin-dependent (type 2) diabetes mellitus and associated neuropathy. This is with a view to developing lead compounds, based on neurotrophic action, for central and peripheral degenerative disorders such as stroke and Alzheimer's disease in addition to the peripheral neuropathy associated with type 2 diabetes mellitus. Here, we address recent advances in the biological action of GLP -1 and its related analogs.
CC Cytology - Animal 02506
Biochemistry studies - Proteins, peptides and amino acids 10064
Biochemistry studies - Carbohydrates 10068
Pathology - Therapy 12512
Metabolism - Metabolic disorders 13020
Cardiovascular system - Blood vessel pathology 14508
Endocrine - General 17002
Endocrine - Pancreas 17008
Endocrine - Neuroendocrinology 17020
Nervous system - Physiology and biochemistry 20504
Nervous system - Pathology 20506
Pharmacology - General 22002
IT Major Concepts
Endocrine System (Chemical Coordination and Homeostasis); Nervous System (Neural Coordination); Pharmacology
IT Parts, Structures, & Systems of Organisms
beta cells: endocrine system; neuronal cells: nervous system
IT Diseases
Alzheimer's disease: behavioral and mental disorders, nervous system disease
Alzheimer Disease (MeSH)
IT Diseases
diabetic neuropathy: endocrine disease/pancreas, metabolic disease, nervous system disease
Diabetic Nephropathies (MeSH)
IT Diseases
stroke: nervous system disease, vascular disease
Cerebrovascular Disorders (MeSH)
IT Diseases
type 2 diabetes mellitus: endocrine disease/pancreas, metabolic disease
Diabetes Mellitus, Non-Insulin-Dependent (MeSH)
IT Chemicals & Biochemicals
glucagon-like peptide-1(7-36)-amide; glucose; insulin
IT Miscellaneous Descriptors
drug development

RN 118549-37-4 (glucagon-like peptide-1(7-36)-amide)
50-99-7Q (glucose)
58367-01-4Q (glucose)
9004-10-8 (insulin)

L33 ANSWER 6 OF 9 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on STN
DUPLICATE 1
ACCESSION NUMBER: 2001:506591 BIOSIS <<LOGINID::20070124>>
DOCUMENT NUMBER: PREV200100506591
TITLE: Urinary excretion of glucagon-like peptide 1 (GLP -1) 7-36 amide in human type 2 (non-insulin-dependent) diabetes mellitus.
AUTHOR(S): Lugari, R. [Reprint author]; Ugolotti, D.; Dei Cas, A.; Barilli, A. L.; Iotti, M.; Marani, B.; Orlandini, A.; Gnudi, A.; Zandomeneghi, R.
CORPORATE SOURCE: Cattedra di Endocrinologia, Via Gramsci 14, 43100, Parma, Italy
endoparm@ipruniv.cce.unipr.it
SOURCE: Hormone and Metabolic Research, (September, 2001) Vol. 33, No. 9, pp. 568-571. print.
CODEN: HMMRA2. ISSN: 0018-5043.
DOCUMENT TYPE: Article
LANGUAGE: English
ENTRY DATE: Entered STN: 31 Oct 2001
Last Updated on STN: 23 Feb 2002
AN 2001:506591 BIOSIS <<LOGINID::20070124>>
DN PREV200100506591
TI Urinary excretion of glucagon-like peptide 1 (GLP-1) 7-36 amide in human type 2 (non-insulin-dependent) diabetes mellitus.
AU Lugari, R. [Reprint author]; Ugolotti, D.; Dei Cas, A.; Barilli, A. L.; Iotti, M.; Marani, B.; Orlandini, A.; Gnudi, A.; Zandomeneghi, R.
CS Cattedra di Endocrinologia, Via Gramsci 14, 43100, Parma, Italy
endoparm@ipruniv.cce.unipr.it
SO Hormone and Metabolic Research, (September, 2001) Vol. 33, No. 9, pp. 568-571. print.
CODEN: HMMRA2. ISSN: 0018-5043.
DT Article
LA English
ED Entered STN: 31 Oct 2001
Last Updated on STN: 23 Feb 2002
AB The urinary excretion of insulinotropic glucagon-like peptide 1 (GLP-1) was investigated as an indicator of renal tubular integrity in 10 healthy subjects and in 3 groups of type 2 diabetic patients with different degrees of urinary albumin excretion rate. No significant difference emerged between the groups with respect to age of

the patients, known duration of diabetes, metabolic control, BMI, or residual beta-cell pancreatic function. Endogenous creatinine clearance was significantly reduced under conditions of overt diabetic nephropathy, compared with normo and microalbuminuric patients ($p<0.01$). Urinary excretion of GLP-1 was significantly higher in normoalbuminuric patients compared to controls (490.4+-211.5 vs. 275.5+-132.1 pg/min; $p<0.05$), with further increase under incipient diabetic nephropathy conditions (648.6+-305 pg/min; $p<0.01$). No significant difference resulted, in contrast, between macroproteinuric patients and non-diabetic subjects. Taking all patients examined into account, a significant positive relationship emerged between urinary GLP-1 and creatinine clearance ($p=0.004$). In conclusion, an early tubular impairment in type 2 diabetes would occur before the onset of glomerular permeability alterations. The tubular dysfunction seems to evolve with the development of persistent microalbuminuria. Finally, the advanced tubular involvement, in terms of urinary GLP1 excretion, under overt diabetic nephropathy conditions would be masked by severe concomitant glomerular damage with the coexistence of both alterations resulting in a peptide excretion similar to control subjects.
CC Biochemistry studies - Proteins, peptides and amino acids 10064
Metabolism - Metabolic disorders 13020
Urinary system - Pathology 15506
Endocrine - General 17002
Endocrine - Pancreas 17008
IT Major Concepts
Clinical Endocrinology (Human Medicine, Medical Sciences); Nephrology (Human Medicine, Medical Sciences)
IT Diseases
diabetic nephropathy: endocrine disease/pancreas, metabolic disease, urologic disease
Diabetic Nephropathies (MeSH)
IT Diseases
type 2 diabetes mellitus: endocrine disease/pancreas, metabolic disease, non-insulin-dependent diabetes mellitus
Diabetes Mellitus, Non-Insulin-Dependent (MeSH)
IT Chemicals & Biochemicals
creatinine; glucagon-like peptide 1: renal tubular integrity indicator; glucagon-like peptide 1 7-36 amide [GLP-1 7-36 amide]: urinary excretion
IT Miscellaneous Descriptors
glomerular permeability
ORGN Classifier
Hominidae 86215
Super Taxa
Primates; Mammalia; Vertebrata; Chordata; Animalia
Organism Name
human: patient
Taxa Notes
Animals, Chordates, Humans, Mammals, Primates, Vertebrates
RN 60-27-5 (creatinine)

L33 ANSWER 7 OF 9 EMBASE COPYRIGHT (c) 2007 Elsevier B.V. All rights reserved on STN
ACCESSION NUMBER: 96034762 EMBASE <<LOGINID::20070124>>
DOCUMENT NUMBER: 1996034762
TITLE: Gastric emptying, glucose responses, and insulin secretion after a liquid test meal: Effects of exogenous glucagon-like peptide-1 (GLP-1)-(7-36) amide in type 2 (noninsulin-dependent) diabetic patients.
AUTHOR: Willms B.; Werner J.; Holst J.J.; Orskov C.; Creutzfeldt W.; Nauck M.A.
CORPORATE SOURCE: Department of Medicine, Ruhr-University Bochum, Knappschafts-Krankenhaus, In der Schornau 23-25,44892 Bochum, Germany
SOURCE: Journal of Clinical Endocrinology and Metabolism, (1996) Vol. 81, No. 1, pp. 327-332. .
ISSN: 0021-972X CODEN: JCEMAZ
COUNTRY: United States
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 003 Endocrinology
037 Drug Literature Index
LANGUAGE: English
SUMMARY LANGUAGE: English
ENTRY DATE: Entered STN: 20 Feb 1996
Last Updated on STN: 20 Feb 1996
AN 96034762 EMBASE <<LOGINID::20070124>>
DN 1996034762
TI Gastric emptying, glucose responses, and insulin secretion after a liquid test meal: Effects of exogenous glucagon-like peptide-1 (GLP-1)-(7-36) amide in type 2 (noninsulin-dependent) diabetic patients.
AU Willms B.; Werner J.; Holst J.J.; Orskov C.; Creutzfeldt W.; Nauck M.A.
CS Department of Medicine, Ruhr-University Bochum, Knappschafts-Krankenhaus, In der Schornau 23-25,44892 Bochum, Germany
SO Journal of Clinical Endocrinology and Metabolism, (1996) Vol. 81, No. 1, pp. 327-332. .
ISSN: 0021-972X CODEN: JCEMAZ
CY United States
DT Journal; Article
FS 003 Endocrinology
037 Drug Literature Index

LA English
SL English
ED Entered STN: 20 Feb 1996
Last Updated on STN: 20 Feb 1996
AB The aim of the study was to investigate whether inhibition of gastric emptying of meals plays a role in the mechanism of the blood glucose-lowering action of glucagon-like peptide-1-(7-36) amide [GLP-1-(7-36) amide] in type 2 diabetes. Eight poorly controlled type 2 diabetic patients (age, 58 ± 6 yr; body mass index, 30.0 ± 5.2 kg/m2; hemoglobin A(1c), 10.5 ± 1.2%) were studied in the fasting state (plasma glucose, 11.1 ± 1.1 mmol/L). A liquid meal of 400 mL containing 8% amino acids and 50 g sucrose (327 Kcal) was administered at time zero by a nasogastric tube. Gastric volume was determined by a dye dilution technique using phenol red. In randomized order, GLP-1-(7-36) amide (1.2 pmol/kg · min; Saxon Biochemicals) or placebo (0.9% NaCl with 1% human serum albumin) was infused between -30 and 240 min. In the control experiment, gastric emptying was completed within 120 min, and plasma glucose, insulin, C-peptide, GLP-1-(7-36) amide, and glucagon concentrations transiently increased. With exogenous GLP-1-(7-36) amide (plasma level, .apprx.70 pmol/L), gastric volume remained constant over the period it was measured (120 min; $P < 0.0001$ vs. placebo), and plasma glucose fell to normal fasting values (5.4 ± 0.7 mmol/L) within 3-4 h, whereas insulin was stimulated in most, but not all, patients, and glucagon remained at the basal level or was slightly suppressed. In conclusion, GLP-1-(7-36) amide inhibits gastric emptying in type 2 diabetic patients. Together with the stimulation of insulin and the inhibition of glucagon secretion, this effect probably contributes to the blood glucose-lowering action of GLP-1-(7-36) amide in type 2-diabetic patients when studied after meal ingestion. At the degree observed, inhibition of gastric emptying, however, must be overcome by tachyphylaxis, reduction in dose, or pharmacological interventions so as not to interfere with the therapeutic use of GLP-1-(7-36) amide in type 2 diabetic patients.
CT Medical Descriptors:
*insulin release
*non insulin dependent diabetes mellitus: DT, drug therapy
*non insulin dependent diabetes mellitus: TH, therapy
*stomach emptying
adult
aged
article
clinical article
clinical trial
controlled study
diabetic angiopathy: CO, complication
diabetic diet
diabetic nephropathy: CO, complication
diabetic neuropathy: CO, complication
diabetic retinopathy
drug effect
drug mechanism
female
glucagon release
glucose blood level
hormone inhibition
human
hypertension: DT, drug therapy
intravenous drug administration
male
postprandial state
priority journal
randomized controlled trial
Drug Descriptors:
*glucagon like peptide 1 [7-36] amide: CM, drug comparison
*glucagon like peptide 1 [7-36] amide: DT, drug therapy
*glucagon like peptide 1 [7-36] amide: PD, pharmacology
*glucagon like peptide 1 [7-36] amide: CT, clinical trial
*glucose: EC, endogenous compound
*insulin: EC, endogenous compound
acarbose: DT, drug therapy
captopril plus hydrochlorothiazide: DT, drug therapy
glibenclamide: DT, drug therapy
isosorbide dinitrate: DT, drug therapy
metformin: DT, drug therapy
metoprolol: DT, drug therapy
nifedipine: DT, drug therapy
placebo: CM, drug comparison
RN (glucagon like peptide 1 [7-36] amide) 119637-73-9; (glucose) 50-99-7, 84778-64-3; (insulin) 9004-10-8; (acarbose) 56180-94-0; (glibenclamide) 10238-21-8; (isosorbide dinitrate) 87-33-2; (metformin) 1115-70-4, 657-24-9; (metoprolol) 37350-58-6; (nifedipine) 21829-25-4
CO Saxon (Germany)

L33 ANSWER 8 OF 9 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on STN
DUPLICATE 2
ACCESSION NUMBER: 1993:408694 BIOSIS <<LOGINID::20070124>>
DOCUMENT NUMBER: PREV199396074419
TITLE: Preserved incretin effect in type 1 diabetic patients with end-stage nephropathy treated by combined heterotopic pancreas and kidney transplantation.
AUTHOR(S): Nauck, M. A. [Reprint author]; Buesing, M.; Orskov, C.; Siegel, E. G.; Talartschik, J.; Baartz, A.; Baartz, T.; Hopt, U. T.; Becker, H.-D.; Creutzfeldt, W.
CORPORATE SOURCE: Div. Gastroenterol. and Endocrinol., Dep. Med., Georg August Univ., Robert-Koch-Strasse 40, W-3400 Goettingen, Germany
SOURCE: Acta Diabetologica, (1993) Vol. 30, No. 1, pp. 39-45.
CODEN: ACDAEZ. ISSN: 0940-5429.
DOCUMENT TYPE: Article
LANGUAGE: English
ENTRY DATE: Entered STN: 8 Sep 1993
Last Updated on STN: 3 Jan 1995
AN 1993:408694 BIOSIS <<LOGINID::20070124>>
DN PREV199396074419
TI Preserved incretin effect in type 1 diabetic patients with end-stage nephropathy treated by combined heterotopic pancreas and kidney transplantation.
AU Nauck, M. A. [Reprint author]; Buesing, M.; Orskov, C.; Siegel, E. G.; Talartschik, J.; Baartz, A.; Baartz, T.; Hopt, U. T.; Becker, H.-D.; Creutzfeldt, W.
CS Div. Gastroenterol. and Endocrinol., Dep. Med., Georg August Univ., Robert-Koch-Strasse 40, W-3400 Goettingen, Germany
SO Acta Diabetologica, (1993) Vol. 30, No. 1, pp. 39-45.
CODEN: ACDAEZ. ISSN: 0940-5429.
DT Article
LA English
ED Entered STN: 8 Sep 1993
Last Updated on STN: 3 Jan 1995
AB Insulin secretion is stimulated better by oral than by intravenous glucose (incretin effect). The contribution of the autonomic nervous system to the incretin effect after oral glucose in humans is unclear. We therefore examined nine type 1 diabetic (insulin-dependent) patients with end-stage nephropathy, studied after combined heterotopic pancreas and kidney transplantation, and 7 non-diabetic kidney recipients (matched for creatinine clearance and immunosuppressive medication). The release of gastric inhibitory polypeptide (GIP) and glucagon-like peptide 1 (GLP-1) immunoreactivity and B cell secretory responses (IR insulin and C-peptide) to oral (50 g) and "isoglycaemic" intravenous glucose (identical glycaemic profile) were measured by radioimmunoassay. The difference in B cell responses between the two tests represents the contribution of the enteroinsular axis to the response after oral glucose (incretin effect). Insulin responses after the oral glucose challenge were similar in the two patient groups despite systemic venous drainage of the pancreas graft in the pancreas-kidney-transplanted group. In both groups GIP and GLP-1 increased after oral but not after intravenous glucose, and B cell secretory responses were significantly smaller (by 55.2 +- 7.7% and 46.5 +- 12.5%, respectively) with "isoglycaemic" intravenous glucose infusions. The lack of reduction in the incretin effect in pancreas-kidney-transplanted patients, whose functioning pancreas is denervated, indicates a lesser role for the nervous system and a more important contribution of circulating incretin hormones in mediating the enteroinsular axis in man.
CC Biochemistry studies - Proteins, peptides and amino acids 10064
Biochemistry studies - Carbohydrates 10068
Anatomy and Histology - Surgery 11105
Anatomy and Histology - Regeneration and transplantation 11107
Pathology - Therapy 12512
Metabolism - Carbohydrates 13004
Metabolism - Proteins, peptides and amino acids 13012
Metabolism - Metabolic disorders 13020
Digestive system - General and methods 14001
Digestive system - Pathology 14006
Urinary system - General and methods 15501
Urinary system - Pathology 15506
Endocrine - Pancreas 17008
IT Major Concepts
Endocrine System (Chemical Coordination and Homeostasis); Gastroenterology (Human Medicine, Medical Sciences); Metabolism; Physiology; Surgery (Medical Sciences); Urology (Human Medicine, Medical Sciences)
IT Chemicals & Biochemicals
INCRETIN; GLUCAGON; INSULIN
IT Miscellaneous Descriptors
ANTIDIABETIC-DRUG; DIABETIC NEUROPATHY; ENZYME INHIBITOR-DRUG
ORGN Classifier
Hominidae 86215
Super Taxa
Primates; Mammalia; Vertebrata; Chordata; Animalia
Organism Name
Hominidae
Taxa Notes
Animals, Chordates, Humans, Mammals, Primates, Vertebrates
RN 54241-84-8 (INCRETIN)
9007-92-5 (GLUCAGON)
9004-10-8 (INSULIN)

L33 ANSWER 9 OF 9 MEDLINE on STN
ACCESSION NUMBER: 92288534 MEDLINE <<LOGINID::20070124>>
DOCUMENT NUMBER: PubMed ID: 1600330

TITLE: Basal and nutrient-stimulated pancreatic and gastrointestinal hormone concentrations in type-1-diabetic patients after successful combined pancreas and kidney transplantation.
AUTHOR: Nauck M A; Busing M; Orskov C; Siegel E G; Talartschik J; Baartz A; Baartz T; Holzer H; Hopt U T; Ebert R; +
CORPORATE SOURCE: Abteilung Gastroenterologie und Endokrinologie, Georg-August-Universitat, Gottingen.
SOURCE: The Clinical investigator, (1992 Jan) Vol. 70, No. 1, pp. 40-8.
Journal code: 9207154. ISSN: 0941-0198.
PUB. COUNTRY: GERMANY: Germany, Federal Republic of
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199207
ENTRY DATE: Entered STN: 24 Jul 1992
Last Updated on STN: 24 Jul 1992
Entered Medline: 13 Jul 1992
AN 92288534 MEDLINE <<LOGINID::20070124>>
DN PubMed ID: 1600330
TI Basal and nutrient-stimulated pancreatic and gastrointestinal hormone concentrations in type-1-diabetic patients after successful combined pancreas and kidney transplantation.
AU Nauck M A; Busing M; Orskov C; Siegel E G; Talartschik J; Baartz A; Baartz T; Holzer H; Hopt U T; Ebert R; +
CS Abteilung Gastroenterologie und Endokrinologie, Georg-August-Universitat, Gottingen.
SO The Clinical investigator, (1992 Jan) Vol. 70, No. 1, pp. 40-8.
Journal code: 9207154. ISSN: 0941-0198.
CY GERMANY: Germany, Federal Republic of
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 199207
ED Entered STN: 24 Jul 1992
Last Updated on STN: 24 Jul 1992
Entered Medline: 13 Jul 1992
AB The secretion of pancreatic and gastrointestinal hormones in the basal state and after nutrient stimuli (50 g glucose, 50 g protein, or 30 g triglyceride administered on separate occasions) was assessed in ten previously type-1-diabetic patients after successful combined kidney and pancreas transplantation (systemic venous drainage). Fasting values were compared to matched non-diabetic kidney-transplanted patients and related to kidney function (endogenous creatinine clearance) and to the type and dosage of immunosuppressive medication. In the fasting state, only IR insulin concentrations were higher in pancreas-kidney-transplanted patients (by 88%; P = 0.001) than in the kidney graft recipients. There were significant inverse correlations of plasma C-peptide, GIP, and gastrin immunoreactivity to endogenous creatinine clearance (kidney function). In response to nutrients, insulin secretion (IR insulin, C-peptide) was significantly stimulated by glucose, and - to a lesser degree - also by protein. Pancreatic glucagon was suppressed by glucose and stimulated by protein ingestion. GIP was raised after glucose and triglyceride more than after protein (P = 0.0003). GLP-1 immunoreactivity was stimulated by all nutrients, with a tendency towards higher responses to protein and fat (P = 0.06). Gastrin was mainly raised by protein. In conclusion, the overall pattern of pancreatic and gastrointestinal hormone release is normal in patients after combined pancreas-kidney-transplantation, but there are some peculiarities due to (a) systemic venous drainage of the pancreas graft (elevated fasting IR insulin) and (b) impaired kidney function (negative correlation of fasting plasma values to endogenous creatinine clearance for C-peptide, GIP, and gastrin).(ABSTRACT TRUNCATED AT 250 WORDS)
CT Check Tags: Female; Male
Adult
Blood Glucose: ME, metabolism
Diabetes Mellitus, Type 1: BL, blood
*Diabetes Mellitus, Type 1: SU, surgery
Diabetic Nephropathies: BL, blood
*Diabetic Nephropathies: SU, surgery
*Gastrointestinal Hormones: BL, blood
Humans
Kidney Function Tests
*Kidney Transplantation: PH, physiology
Middle Aged
*Pancreas Transplantation: PH, physiology
Pancreatic Function Tests
*Pancreatic Hormones: BL, blood
Research Support, Non-U.S. Gov't
CN 0 (Blood Glucose); 0 (Gastrointestinal Hormones); 0 (Pancreatic Hormones)

D Ibib all L34 1-9

L34 ANSWER 1 OF 9 CAPLUS COPYRIGHT 2007 ACS on STN
ACCESSION NUMBER: 2004:533962 CAPLUS <<LOGINID::20070124>>
DOCUMENT NUMBER: 141:82335
TITLE: Human glucagon-like-peptide-1 mimics and their antidiabetic effects
INVENTOR(S): Natarajan, Sesha Iyer; Mapelli, Claudio; Bastos, Margarita M.; Bernatowicz, Michael; Lee, Ving; Ewing, William R.

PATENT ASSIGNEE(S): USA
SOURCE: U.S. Pat. Appl. Publ., 73 pp., Cont.-in-part of U.S. Ser. No. 273,975.
CODEN: USXXCO
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 2
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
US 2004127423	A1	20040701	US 2003-419399	20030421
US 2003195157	A1	20031016	US 2002-273975	20021018 <--
WO 2004094461	A2	20041104	WO 2004-US12374	20040421
WO 2004094461	A3	20050915		

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW
RW: BW, GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG

EP 1615653	A2	20060118	EP 2004-760098	20040421

R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR, BG, CZ, EE, HU, PL, SK, HR

PRIORITY APPLN. INFO.: US 2001-342015P P 20011018
US 2002-273975 A2 20021018
US 2003-419399 A 20030421
WO 2004-US12374 W 20040421

AN 2004:533962 CAPLUS <<LOGINID::20070124>>
DN 141:82335
ED Entered STN: 02 Jul 2004
TI Human glucagon-like-peptide-1 mimics and their antidiabetic effects
IN Natarajan, Sesha Iyer; Mapelli, Claudio; Bastos, Margarita M.; Bernatowicz, Michael; Lee, Ving; Ewing, William R.
PA USA
SO U.S. Pat. Appl. Publ., 73 pp., Cont.-in-part of U.S. Ser. No. 273,975.
CODEN: USXXCO
DT Patent
LA English
IC ICM A61K038-10
ICS C07K007-08
INCL 514015000; 530328000
CC 1-10 (Pharmacology)
Section cross-reference(s): 2, 34, 63
FAN.CNT 2

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI US 2004127423	A1	20040701	US 2003-419399	20030421
US 2003195157	A1	20031016	US 2002-273975	20021018 <--
WO 2004094461	A2	20041104	WO 2004-US12374	20040421
WO 2004094461	A3	20050915		

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW
RW: BW, GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG

EP 1615653	A2	20060118	EP 2004-760098	20040421

R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR, BG, CZ, EE, HU, PL, SK, HR

PRAI US 2001-342015P P 20011018
US 2002-273975 A2 20021018
US 2003-419399 A 20030421
WO 2004-US12374 W 20040421

CLASS

PATENT NO.	CLASS	PATENT FAMILY CLASSIFICATION CODES
US 2004127423	ICM	A61K038-10
	ICS	C07K007-08
	INCL	514015000; 530328000
	IPCI	A61K0038-10 [ICM,7]; C07K0007-08 [ICS,7]; C07K0007-00 [ICS,7,C*]
	IPCR	A61K0038-00 [N,C*]; A61K0038-00 [N,A]; C07K0014-435 [I,C*]; C07K0014-605 [I,A]
	NCL	514/015.000; 530/328.000
	ECLA	C07K014/605
US 2003195157	IPCI	A61K0038-10 [ICM,7]; A61K0038-08 [ICS,7]; C07K0007-08 [ICS,7]; C07K0007-06 [ICS,7]; C07K0007-00 [ICS,7,C*]
	IPCR	A61K0038-00 [N,C*]; A61K0038-00 [N,A]; C07K0014-435 [I,C*]; C07K0014-605 [I,A]
	NCL	514/016.000; 514/017.000; 530/328.000; 530/329.000
	ECLA	C07K014/605

WO 2004094461 IPCI C07K [ICM,7]
IPCR A61K0038-00 [I,C*]; A61K0038-00 [I,A]; A61K0038-02 [I,C*]; A61K0038-02 [I,A]; A61K0038-08 [I,C*]; A61K0038-08 [I,A]; A61K0038-10 [I,C*]; A61K0038-10 [I,A]; C07K [I,S]; C07K0007-00 [I,C*]; C07K0007-02 [I,A]; C07K0007-04 [I,A]; C07K0007-08 [I,A]
EP 1615653 IPCI A61K0038-00 [ICM,7]; A61K0038-02 [ICS,7]; A61K0038-10 [ICS,7]; A61K0038-08 [ICS,7]; C07K0007-02 [ICS,7]; C07K0007-04 [ICS,7]; C07K0007-08 [ICS,7]; C07K0007-00 [ICS,7,C*]
IPCR A61K0038-00 [I,C*]; A61K0038-00 [I,A]; A61K0038-02 [I,C*]; A61K0038-02 [I,A]; A61K0038-08 [I,C*]; A61K0038-08 [I,A]; A61K0038-10 [I,C*]; A61K0038-10 [I,A]; C07K [I,S]; C07K0007-00 [I,C*]; C07K0007-02 [I,A]; C07K0007-04 [I,A]; C07K0007-08 [I,A]

AB The invention discloses human glucagon-like peptide-1 (GLP-1) peptide mimics that mimic the biol. activity of the native GLP-1 peptide and thus are useful for the treatment or prevention of diseases or disorders associated with GLP activity. Further, the invention provides novel, chemical modified peptides that not only stimulate insulin secretion in type II diabetics, but also produce other beneficial insulinotropic responses. These synthetic peptide GLP -1 mimics exhibit increased stability to proteolytic cleavage making them ideal therapeutic candidates for oral or parenteral administration.

ST human glucagon peptide mimic prepn diabetes antidiabetic insulin stability

IT Proteins
RL: PAC (Pharmacological activity); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(ALBP (adipocyte lipid-binding protein); human glucagon-like-peptide-1 mimics and their antidiabetic effects)

IT Lipoprotein receptors
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(LDL; human glucagon-like-peptide-1 mimics and their antidiabetic effects)

IT Proteins
RL: PAC (Pharmacological activity); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(MTP (microsomal triglyceride-exchanging protein), inhibitors; human glucagon-like-peptide-1 mimics and their antidiabetic effects)

IT Antiarteriosclerotics
(antiatherosclerotics; human glucagon-like-peptide-1 mimics and their antidiabetic effects)

IT Drug delivery systems
(capsules; human glucagon-like-peptide-1 mimics and their antidiabetic effects)

IT Proteins
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(cholesterol ester-exchanging; human glucagon-like-peptide-1 mimics and their antidiabetic effects)

IT Kidney, disease
(diabetic nephropathy; human glucagon-like-peptide-1 mimics and their antidiabetic effects)

IT Nerve, disease
(diabetic neuropathy; human glucagon-like-peptide-1 mimics and their antidiabetic effects)

IT Eye, disease
(diabetic retinopathy; human glucagon-like-peptide-1 mimics and their antidiabetic effects)

IT Transport proteins
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(dopamine transporter; human glucagon-like-peptide-1 mimics and their antidiabetic effects)

IT 5-HT reuptake inhibitors
Antihypertensives
Antiobesity agents
Appetite depressants
Atherosclerosis
Diabetes mellitus
Human
Hyperglycemia
Hypertension
Hypertriglyceridemia
Hypolipemic agents
Obesity
Signal transduction, biological
Wound healing
b3-Adrenoceptor agonists
(human glucagon-like-peptide-1 mimics and their antidiabetic effects)

IT Fatty acids, biological studies
Glucagon-like peptide-1 receptors
Hyperlipidemia
Thyroid hormone receptors
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(human glucagon-like-peptide-1 mimics and their antidiabetic effects)

IT Peptides, biological studies
RL: PAC (Pharmacological activity); SPN (Synthetic preparation); THU (Therapeutic use); BIOL (Biological study); PREP (Preparation); USES (Uses)
(human glucagon-like-peptide-1 mimics and their antidiabetic effects)

IT Sulfonylureas
RL: PAC (Pharmacological activity); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(human glucagon-like-peptide-1 mimics and their antidiabetic effects)

IT Drug delivery systems
(injections; human glucagon-like-peptide-1 mimics and their antidiabetic effects)

IT Metabolic disorders
(metabolic syndrome X; human glucagon-like-peptide-1 mimics and their antidiabetic effects)

IT Drug delivery systems
(microparticles; human glucagon-like-peptide-1 mimics and their antidiabetic effects)

IT Diabetes mellitus
(non-insulin-dependent; human glucagon-like-peptide-1 mimics and their antidiabetic effects)

IT Antidiabetic agents
Drug delivery systems
(oral; human glucagon-like-peptide-1 mimics and their antidiabetic effects)

IT Drug delivery systems
(suspensions; human glucagon-like-peptide-1 mimics and their antidiabetic effects)

IT Drug delivery systems
(tablets; human glucagon-like-peptide-1 mimics and their antidiabetic effects)

IT Peroxisome proliferator-activated receptors
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(a; human glucagon-like-peptide-1 mimics and their antidiabetic effects)

IT Peroxisome proliferator-activated receptors
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(g; human glucagon-like-peptide-1 mimics and their antidiabetic effects)

IT 51-61-6, Dopamine, biological studies 56-81-5, Glycerol, biological studies 9001-62-1, Lipase 9028-35-7, 3-Hydroxy-3-methylglutaryl CoA reductase 9029-60-1, Lipoxygenase 9033-06-1, Glucosidase 9077-14-9, Squalene synthetase 63551-74-6, Lipoxygenase 90002-36-1, 2-Ethylphenyl boronic acid
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(human glucagon-like-peptide-1 mimics and their antidiabetic effects)

IT 516514-32-2P 516514-38-8P 516514-43-5P 516514-47-9P 516514-52-6P 516514-55-9P 516514-58-2P 516514-61-7P 516514-64-0P 516514-68-4P 516514-72-0P 516514-75-3P 516514-78-6P 516514-81-1P 516514-84-4P 516514-87-7P 516514-91-3P 516514-95-7P 516514-99-1P 516515-03-0P 516515-06-3P 516515-09-6P 516515-14-3P 516515-18-7P 516515-22-3P 516515-26-7P 516515-30-3P 516515-34-7P 516515-38-1P 516515-42-7P 516515-46-1P 516515-50-7P 516515-55-2P 516515-59-6P 516515-63-2P 516515-68-7P 516515-72-3P 516515-76-7P 516515-80-3P 516515-84-7P 516515-88-1P 516515-92-7P 516515-96-1P 516516-01-1P 516516-06-6P 516516-10-2P 516516-14-6P 516516-18-0P 516516-22-6P 516516-26-0P 516516-31-7P 516516-35-1P 516516-39-5P 516516-44-2P 516516-50-0P 516516-55-5P 516516-60-2P 516516-64-6P 516516-68-0P 516516-72-6P 516516-76-0P 516516-80-6P 516516-85-1P 516516-87-3P 516516-91-9P 516516-95-3P 516516-98-6P 516517-02-5P 516517-06-9P 516517-10-5P 516517-14-9P 516517-17-2P 516517-22-9P 516517-26-3P 516517-30-9P 516517-33-2P 516517-37-6P 516517-41-2P 516517-45-6P 516517-50-3P 516517-54-7P 516517-59-2P 516517-63-8P 516517-67-2P 516517-71-8P 516517-75-2P 516517-79-6P 516517-82-1P 516517-85-4P 516517-88-7P 516517-91-2P 516517-96-7P 516518-00-6P 516518-04-0P 516518-08-4P 516518-11-9P 516518-15-3P 516518-19-7P 516518-22-2P 516518-26-6P 516518-30-2P 516518-33-5P 516518-35-7P 516518-39-1P 516518-42-6P 516518-46-0P 516518-48-2P 516518-51-7P 516518-54-0P 516518-57-3P 516518-59-5P 516518-61-9P 516518-64-2P 516518-66-4P 516518-69-7P 516518-73-3P 516518-78-8P 516518-83-5P 516518-88-0P 516518-92-6P 516518-96-0P 516519-00-9P 516519-04-3P 516519-09-8P 516519-12-3P 516519-15-6P 516519-18-9P 516519-21-4P 516519-24-7P 516519-27-0P 516519-32-7P 516519-37-2P 516519-40-7P 516519-45-2P 516519-50-9P 516519-54-3P 516519-59-8P 516519-63-4P 516519-67-8P 516519-72-5P 516519-77-0P 516519-82-7P 516519-87-2P 516519-91-8P 516519-95-2P 516519-99-6P 516520-03-9P 516520-09-5P 516520-13-1P 516520-17-5P 516520-22-2P 516520-26-6P 516520-29-9P 516520-33-5P 516520-36-8P 516520-39-1P 516520-42-6P 516520-45-9P 516520-47-1P 516520-49-3P 516520-52-8P 516520-54-0P 516520-55-1P 516520-57-3P 516520-59-5P 516520-61-9P 516520-63-1P 516520-66-4P 516520-68-6P 516520-70-0P 516520-72-2P 516520-74-4P 516520-75-5P 516520-77-7P 516520-79-9P 516520-81-3P 516520-82-4P 516520-84-6P 516520-86-8P 516520-87-9P 516520-89-1P 516520-91-5P 516520-93-7P 516520-95-9P 516520-97-1P 516520-99-3P 516521-01-0P 516521-03-2P 516521-05-4P 516521-07-6P 516521-08-7P 516521-09-8P 516521-10-1P 516521-12-3P 516521-13-4P 516521-14-5P 516521-16-7P 516521-18-9P 516521-19-0P 516521-21-4P 516521-22-5P 516521-23-6P 516521-24-7P 516521-25-8P 516521-26-9P 516521-27-0P 516521-28-1P 516521-29-2P 516521-30-5P 516521-31-6P 516521-32-7P 516521-33-8P 516521-34-9P 516521-35-0P 516521-36-1P 516521-37-2P 516521-38-3P 516521-39-4P 516521-40-7P 516521-41-8P 516521-42-9P 516521-43-0P 516521-44-1P 516521-45-2P 516521-53-2P 516521-54-3P 516521-55-4P 713497-71-3P 713497-72-4P 713497-73-5P 713497-74-6P 713497-75-7P 713497-77-9P
RL: PAC (Pharmacological activity); PRP (Properties); SPN (Synthetic preparation); THU (Therapeutic use); BIOL (Biological study); PREP (Preparation); USES (Uses)
(human glucagon-like-peptide-1 mimics and their antidiabetic effects)

IT 713497-79-1P 713497-81-5P 713497-83-7P 713497-85-9P

RL: PAC (Pharmacological activity); PRP (Properties); SPN (Synthetic preparation); THU (Therapeutic use); BIOL (Biological study); PREP (Preparation); USES (Uses)
(human glucagon-like-peptide-1 mimics and their antidiabetic effects)

IT 51-64-9, Dexamphet-amine 56-03-1, Biguanide 94-20-2, Chloropropamide 122-09-8, Phentermine 637-07-0, Clofibrate 657-24-9, Metformin 943-45-3D, Fibric acid, derivs. 10238-21-8, Glyburide 14838-15-4, Phenylpropanolamine 21187-98-4, Gliclazide 22232-71-9, Mazindol 25812-30-0, Gemfibrozil 29094-61-9, Glipizide 49562-28-9, Fenofibrate 54870-28-9, Meglitinide 56180-94-0, Acarbose 72432-03-2, Miglitol 75330-75-5, Lovastatin 79902-63-9, Simvastatin 81093-37-0, Pravastatin 89750-14-1, Glucagon-like peptide I 93479-97-1, Glimepiride 93957-54-1, Fluvastatin 96829-58-2, Orlistat 97240-79-4, Topiramate 97322-87-7, Troglitazone 105816-04-4, Nateglinide 106650-56-0, Sibutramine 111025-46-8, Pioglitazone 122320-73-4, Rosiglitazone 134523-00-5, Ator-vastatin 135062-02-1, Repaglinide 141750-63-2, Nisvastatin 141758-74-9, AC2993 144288-97-1, TS-962 145599-86-6, Cerivastatin 152755-31-2, LY295427 159183-92-3, L750355 161600-01-7, Isaglitazone 166518-60-1, Avasimibe 170861-63-9, JTT-501 176435-10-2, LY315902 178759-95-0, MD 700 196808-45-4 199113-98-9, NN-2344 199914-96-0, YM-440 213252-19-8, KRP297 244081-42-3, AJ9677 258345-41-4, GW-409544 262352-17-0, CP 529414 282526-98-1, ATL-962 287714-41-4 335149-08-1, L895645 335149-14-9, R-119702 335149-15-0, KAD1129 335149-17-2, AR-HO39242 335149-23-0, NVP-DPP-728A 335149-25-2, CP331648 430433-17-3, Glipyride 444069-80-1, Axokine
RL: PAC (Pharmacological activity); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(human glucagon-like-peptide-1 mimics and their antidiabetic effects)

IT 150-46-9, Triethylborate 358-23-6, Triflic anhydride 1973-22-4, 1-Bromo-2-ethylbenzene 4326-36-7 16419-60-6, O-Tolylboronic acid 82911-69-1 93267-04-0 516521-49-6 713497-86-0
RL: RCT (Reactant); RACT (Reactant or reagent)
(human glucagon-like-peptide-1 mimics and their antidiabetic effects)

IT 112766-18-4P 516521-46-3P 516521-47-4P 516521-48-5P 516521-50-9P 516521-51-0P
RL: RCT (Reactant); SPN (Synthetic preparation); PREP (Preparation); RACT (Reactant or reagent)
(human glucagon-like-peptide-1 mimics and their antidiabetic effects)

IT 713497-87-1P 713497-88-2P
RL: SPN (Synthetic preparation); PREP (Preparation)
(human glucagon-like-peptide-1 mimics and their antidiabetic effects)

IT 9027-63-8, ACAT
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(inhibitors; human glucagon-like-peptide-1 mimics and their antidiabetic effects)

IT 54249-88-6, Dipeptidyl peptidase IV
RL: PAC (Pharmacological activity); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(inhibitors; human glucagon-like-peptide-1 mimics and their antidiabetic effects)

IT 9004-10-8, Insulin, biological studies
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(resistance, hyperinsulinemia; human glucagon-like-peptide-1 mimics and their antidiabetic effects)

L34 ANSWER 2 OF 9 CAPLUS COPYRIGHT 2007 ACS on STN
ACCESSION NUMBER: 2004:157498 CAPLUS <<LOGINID::20070124>>
DOCUMENT NUMBER: 140:199313
TITLE: Preparation of fused pyrrolylcarboxamides as glycogen phosphorylase inhibitors
INVENTOR(S): Daisy, Joe
PATENT ASSIGNEE(S): Pfizer Products Inc., USA
SOURCE: Eur. Pat. Appl., 71 pp.
CODEN: EPXXDW
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 2
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
EP 1391460	A1	20040225	EP 2003-20676	20000918
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, FI, CY				
EP 1088824	A2	20010404	EP 2000-308131	20000918 <--
EP 1088824	A3	20010627		
EP 1088824	B1	20040107		
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO				
US 2002183369	A1	20021205	US 2002-117370	20020405 <--
US 6576653	B2	20030610		
US 2003195361	A1	20031016	US 2003-367002	20030214 <--
US 6828343	B2	20041207		

PRIORITY APPLN. INFO.: US 1999-157148P P 19990930
EP 2000-308131 A3 20000918
US 2000-670759 A3 20000927
US 2002-117370 A3 20020405
OTHER SOURCE(S): MARPAT 140:199313
AN 2004:157498 CAPLUS <<LOGINID::20070124>>
DN 140:199313
ED Entered STN: 26 Feb 2004
TI Preparation of fused pyrrolylcarboxamides as glycogen phosphorylase inhibitors
IN Daisy, Joe
PA Pfizer Products Inc., USA
SO Eur. Pat. Appl., 71 pp.
CODEN: EPXXDW
DT Patent
LA English
IC ICM C07D495-04
ICS C07D491-04; C07D209-52; A61K031-407; A61P003-10; A61P009-10; C07D495-14; C07D333-00; C07D209-00; C07D307-00
CC 28-2 (Heterocyclic Compounds (More Than One Hetero Atom))
Section cross-reference(s): 1, 63
FAN.CNT 2

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI EP 1391460	A1	20040225	EP 2003-20676	20000918
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, FI, CY				
EP 1088824	A2	20010404	EP 2000-308131	20000918 <--
EP 1088824	A3	20010627		
EP 1088824	B1	20040107		
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO				
US 2002183369	A1	20021205	US 2002-117370	20020405 <--
US 6576653	B2	20030610		
US 2003195361	A1	20031016	US 2003-367002	20030214 <--
US 6828343	B2	20041207		

PRAI US 1999-157148P P 19990930
EP 2000-308131 A3 20000918
US 2000-670759 A3 20000927
US 2002-117370 A3 20020405
CLASS

PATENT NO.	CLASS	PATENT FAMILY CLASSIFICATION CODES
EP 1391460	ICM	C07D495-04
	ICS	C07D491-04; C07D209-52; A61K031-407; A61P003-10; A61P009-10; C07D495-14; C07D333-00; C07D209-00; C07D307-00
	IPCI	C07D0495-04 [ICM,7]; C07D0491-04 [ICS,7]; C07D0491-00 [ICS,7,C*]; C07D0209-52 [ICS,7]; A61K0031-407 [ICS,7]; A61P0003-10 [ICS,7]; A61P0003-00 [ICS,7,C*]; A61P0009-10 [ICS,7]; A61P0009-00 [ICS,7,C*]; C07D0495-14 [ICS,7]; C07D0495-00 [ICS,7,C*]; C07D0333-00 [ICS,7]; C07D0209-00 [ICS,7]; C07D0307-00 [ICS,7]
	ECLA	C07D491/04+307B+209B; C07D495/04+333B+209B; C07D495/14+333B+333B+209B
EP 1088824	IPCI	C07D0495-04 [ICM,6]; C07D0491-04 [ICS,6]; C07D0209-52 [ICS,6]; A61K0031-407 [ICS,6]; A61P0003-10 [ICS,6]; A61P0003-00 [ICS,6,C*]; A61P0009-10 [ICS,6]; A61P0009-00 [ICS,6,C*]; C07D0495-04 [ICI,6]; C07D0495-00 [ICI,6,C*]; C07D0333-00 [ICI,6]; C07D0209-00 [ICI,6]; C07D0491-04 [ICI,6]; C07D0491-00 [ICI,6,C*]; C07D0307-00 [ICI,6]; C07D0209-00 [ICI,6]
	IPCR	C07D0491-048 [I,A]; A61K0031-407 [I,C*]; A61K0031-407 [I,A]; A61K0031-427 [I,C*]; A61K0031-427 [I,A]; A61K0031-4523 [I,C*]; A61K0031-454 [I,A]; A61K0031-5375 [I,C*]; A61K0031-5377 [I,A]; A61K0031-695 [I,C*]; A61K0031-695 [I,A]; A61K0038-00 [I,C*]; A61K0038-00 [I,A]; A61K0045-00 [I,C*]; A61K0045-00 [I,A]; A61P0003-00 [I,C*]; A61P0003-06 [I,A]; A61P0003-10 [I,A]; A61P0009-00 [I,C*]; A61P0009-10 [I,A]; A61P0009-12 [I,A]; A61P0027-00 [I,C*]; A61P0027-12 [I,A]; A61P0043-00 [I,C*]; A61P0043-00 [I,A]; C07D0209-00 [I,C*]; C07D0209-52 [I,A]; C07D0491-00 [I,C*]; C07D0491-04 [I,A]; C07D0495-00 [I,C*]; C07D0495-04 [I,A]; C07D0495-14 [I,A]; C07F0007-00 [I,C*]; C07F0007-10 [I,A]; C07K0005-00 [I,C*]; C07K0005-00 [I,A]
	ECLA	C07D209/52; C07D491/04+307B+209B; C07D495/04+333B+209B
US 2002183369	IPCI	C07D0513-22 [ICM,7]; C07D0513-00 [ICM,7,C*]; A61K0031-429 [ICS,7]; A61K0031-424 [ICS,7]; A61K0031-4188 [ICS,7]; A61K0031-4164 [ICS,7,C*]
	IPCR	C07D0209-00 [I,C*]; C07D0209-52 [I,A]; C07D0491-00 [I,C*]; C07D0491-04 [I,A]; C07D0495-00 [I,C*]; C07D0495-04 [I,A]
	NCL	514/367.000; 514/375.000; 514/393.000; 514/412.000; 548/153.000; 548/217.000; 548/303.100; 548/453.000
	ECLA	C07D209/52; C07D491/04+307B+209B; C07D495/04+333B+209B
US 2003195361	IPCI	C07D0513-12 [ICM,7]; C07D0498-02 [ICS,7]; C07D0498-00 [ICS,7,C*]; C07D0513-02 [ICS,7]; C07D0513-00 [ICS,7,C*]; C07D0487-02 [ICS,7]; C07D0487-00 [ICS,7,C*]
	IPCR	C07D0209-00 [I,C*]; C07D0209-52 [I,A]; C07D0491-00 [I,C*]; C07D0491-04 [I,A]; C07D0495-00 [I,C*]; C07D0495-04 [I,A]
	NCL	548/153.000; 548/218.000; 548/303.100; 548/453.000
	ECLA	C07D209/52; C07D491/04+307B+209B; C07D495/04+333B+209B

OS MARPAT 140:199313
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AB Title compds. [I; Q = substituted aryl, heteroaryl; Z, X = C, CH, CH2, N, O, S; X1 = NRa, CH2, O , S; dotted lines = bond, null; both dotted lines are not simultaneously bonds; R1 = H, halo, alkoxy, alkylthio, alkyl, CF3, NH2, alkylamino, dialkylamino, NO2, CN, CO2H, carboxyalkyl, alkenyl, alkynyl; Ra, Rb = H, alkyl; Y = CH(OH), null; R2R3 = atoms to form a 5-6 membered ring containing 0-3 heteroatoms and 0-2 double bonds; R4 = COA; A = NRdRd, NRaCH2CH2ORa, N-heterocyclyl; Rd = H, alkyl, alkoxy, aryl, (substituted) aryl, heteroaryl; Rc = H, CO2Ra, ORa, SRa, NRaRa; n = 1-3], were prepared for treatment of diabetes, insulin resistance, diabetic neuropathy, diabetic nephropathy, diabetic retinopathy, cataracts, hyperglycemia, hypercholesterolemia, hypertension, hyperinsulinemia, hyperlipidemia, atherosclerosis, or tissue ischemia (no data). Thus, 6H-thieno[2,3-b]pyrrole-5-carboxylic acid and (3S)-amino-1-((3R,4S)-dihydroxypyrrolidin-1-yl)-(2R)-hydroxy-4-phenylbutan-1-one were coupled using 4-(dimethylamino)pyridine, 1-hydroxybenzotriazole hydrate and 1-(3-dimethylaminopropyl)-3-ethylcarbodiimide hydrochloride in CH2Cl2/DMF to give 6H-thieno[2,3-b]pyrrole-5-carboxylic acid [(1S)-benzyl-3-((3R,4S)-dihydroxypyrrolidin-1-yl)-(2R)-hydroxy-3-oxopropyl]amide.

ST pyrrolylcarboxamide fused prepn glycogen phosphorylase inhibitor; thienopyrrolecarboxamide prepn antidiabetic; diabetes insulin resistance diabetic neuropathy treatment fused pyrrolecarboxamide; diabetic nephropathy retinopathy cataract hyperglycemia hypercholesterolemia hypertension treatment pyrrolecarboxamide; hyperinsulinemia hyperlipidemia atherosclerosis tissue ischemia treatment fused pyrrolecarboxamide

IT Ischemia
(cardiac, treatment; preparation of fused pyrrolylcarboxamides as glycogen phosphorylase inhibitors)

IT Antiobesity agents
a2-Adrenoceptor antagonists
b-Adrenoceptor agonists
(coadministration; preparation of fused pyrrolylcarboxamides as glycogen phosphorylase inhibitors)

IT Sulfonylureas
RL: THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(coadministration; preparation of fused pyrrolylcarboxamides as glycogen phosphorylase inhibitors)

IT Kidney, disease
(diabetic nephropathy, treatment; preparation of fused pyrrolylcarboxamides as glycogen phosphorylase inhibitors)

IT Nerve, disease
(diabetic neuropathy, treatment; preparation of fused pyrrolylcarboxamides as glycogen phosphorylase inhibitors)

IT Eye, disease
(diabetic retinopathy, treatment; preparation of fused pyrrolylcarboxamides as glycogen phosphorylase inhibitors)

IT Antioxidants
(fatty acid oxidation inhibitors coadministration; preparation of fused pyrrolylcarboxamides as glycogen phosphorylase inhibitors)

IT Gluconeogenesis
(inhibitors coadministration; preparation of fused pyrrolylcarboxamides as glycogen phosphorylase inhibitors)

IT Heart, disease
(ischemia, treatment; preparation of fused pyrrolylcarboxamides as glycogen phosphorylase inhibitors)

IT Anti-ischemic agents
Anticholesteremic agents
Antidiabetic agents
Antihypertensives
Drug delivery systems
Human
Hypolipemic agents
(preparation of fused pyrrolylcarboxamides as glycogen phosphorylase inhibitors)

IT Atherosclerosis
Cataract
Diabetes mellitus
Hypercholesterolemia
Hyperglycemia
Hypertension
Hypertriglyceridemia
Ischemia
(treatment; preparation of fused pyrrolylcarboxamides as glycogen phosphorylase inhibitors)

IT Hyperlipidemia
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(treatment; preparation of fused pyrrolylcarboxamides as glycogen phosphorylase inhibitors)

IT Peroxisome proliferator-activated receptors
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(g, PPAR-g agonists coadministration; preparation of fused pyrrolylcarboxamides as glycogen phosphorylase inhibitors)

IT 9007-92-5, Glucagon, biological studies 106602-62-4, Amylin
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(antagonists coadministration; preparation of fused pyrrolylcarboxamides as glycogen phosphorylase inhibitors)

IT 56-03-1D, Biguanide, derivs. 64-77-7, Tolbutamide 94-20-2, Chloropropamide 114-86-3, Phenformin 458-24-2, Fenfluramine 657-24-9, Metformin 692-13-7, Buformin 968-81-0, Acetohexamide 1156-19-0, Tolazamide 2295-31-0D, Thiazolidinedione, derivs. 7440-62-2D, Vanadium, complexes 9004-10-8, Insulin, biological studies 9004-10-8D, Insulin, analogs 10238-21-8, Glibenclamide 12179-36-1D, Pervanadyl (VO(O2)+), complexes 23602-78-0, Benfluorex 28299-33-4D, Imidazoline, derivs. 29094-61-9, Glipizide 37353-31-4, Vanadate 51037-30-0, Acipimox 51110-01-1D, Somatostatin, analogs 54870-28-9, Meglitinide 56180-94-0, Acarbose 66529-17-7, Midaglizole 72432-03-2, Miglitol 74772-77-3, Ciglitazone 75358-37-1, Linogliride 79944-58-4, Idazoxan 80879-63-6, Emiglitate 83480-29-9, Voglibose 86615-96-5, BRL35135 88431-47-4, Clomoxir 89197-32-0, Efaroxan 90505-66-1, Ro 16-8714 90730-96-4, BRL37344 93479-97-1, Glimepiride 97322-87-7, Troglitazone 104343-33-1, MDL-25637 105182-45-4, Fluparoxan 105816-04-4, Nateglinide 106612-94-6, Human GLP-1 -(7-37) 107444-51-9, Rat GLP-1(7-36)amide 109229-58-5, Englitazone 110605-64-6, Isaglidole 111025-46-8, Pioglitazone 115656-32-1, D 7114 122320-73-4, Rosiglitazone 122575-28-4, Naglivan 122830-14-2, Deriglidole 124083-20-1, Etomoxir 127214-23-7, Camiglibose 130714-47-5, WAG 994 133107-64-9, Insulin lispro 135062-02-1, Repaglinide 138908-40-4, CL 316243 141200-24-0, Darglitazone 141758-74-9, AC2993 187887-46-3, Symlin 395214-16-1
RL: THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(coadministration; preparation of fused pyrrolylcarboxamides as glycogen phosphorylase inhibitors)

IT 9001-42-7, a-Glucosidase 9025-82-5, Phosphodiesterase
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(inhibitors coadministration; preparation of fused pyrrolylcarboxamides as glycogen phosphorylase inhibitors)

IT 9035-74-9, Glycogen phosphorylase
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(inhibitors; preparation of fused pyrrolylcarboxamides as glycogen phosphorylase inhibitors)

IT 332098-11-0P 332098-12-1P 332098-13-2P 332098-14-3P 332098-15-4P 332098-16-5P 332098-17-6P 332098-18-7P 332098-19-8P 332098-20-1P 332098-21-2P 332098-22-3P 332098-23-4P 332098-24-5P 332098-25-6P 332098-26-7P 332098-27-8P 332098-28-9P 332098-29-0P 332098-30-3P 332098-31-4P 332098-32-5P 332098-33-6P 332098-34-7P 332098-35-8P 332098-36-9P 332098-37-0P 332098-38-1P 332098-39-2P 332098-40-5P 332098-41-6P 332098-42-7P 332098-43-8P 332098-44-9P 332098-45-0P 332098-46-1P 332098-47-2P 332098-48-3P 332098-49-4P 332098-50-7P 332098-52-9P 332098-54-1P 332098-55-2P 332098-57-4P 332098-59-6P 332098-61-0P 332098-63-2P 332098-65-4P
RL: PAC (Pharmacological activity); SPN (Synthetic preparation); THU (Therapeutic use); BIOL (Biological study); PREP (Preparation); USES (Uses)
(preparation of fused pyrrolylcarboxamides as glycogen phosphorylase inhibitors)

IT 637-81-0, Azidoacetic acid ethyl ester 1066-54-2, (Trimethylsilyl)acetylene 1126-09-6, Piperidine-4-carboxylic acid ethyl ester 4530-18-1, Boc-DL-Phenylalanine 6030-36-0, 4-Methylthiophene-2-carboxaldehyde 7283-96-7, 5-Chlorothiophene-2-carboxaldehyde 13679-70-4, 5-Methyl-2-thiophenecarboxaldehyde 13734-34-4, Boc-L-Phenylalanine 14345-97-2, 2-Chloro-3-methylthiophene 17186-57-1 18791-75-8, 4-Bromothiophene-2-carboxaldehyde 21508-19-0, 5-Chlorofuran-2-carboxaldehyde 21921-76-6, 4-Bromo-2-furaldehyde 24445-35-0 29669-49-6, 5-Fluorothiophene-2-carboxaldehyde 31486-85-8, Thieno[2,3-b]thiophene-2-carboxaldehyde 35357-56-3, 6H-Thieno[2,3-b]pyrrole-5-carboxylic acid ethyl ester 39793-31-2, 4H-Thieno[3,2-b]pyrrole-5-carboxylic acid 51856-25-8, 6H-Thieno[2,3-b]pyrrole-5-carboxylic acid 51856-29-2, 2-Formyl-6H-thieno[2,3-b]pyrrole-5-carboxylic acid ethyl ester 57500-51-3, 4-Chlorothiophene-2-carboxaldehyde 58963-45-4, 2-Formyl-6H-thieno[2,3-b]pyrrole-5-carboxylic acid 59958-27-9, 2-Formyl-4H-thieno[3,2-b]pyrrole-5-carboxylic acid ethyl ester 62023-60-3, (2R,3S)-3-Benzyloxycarbonylamino-2-hydroxy-4-phenylbutyric acid 80709-80-4, 2-Methyl-4H-furo[3,2-b]pyrrole-5-carboxylic acid 80709-83-7, 2-Bromo-4H-furo[3,2-b]pyrrole-5-carboxylic acid ethyl ester 91545-55-0, 2-Bromo-6H-thieno[2,3-b]pyrrole-5-carboxylic acid ethyl ester 105181-72-4, (2R,3S)-3-tert-Butoxycarbonylamino-2-hydroxy-4-phenylbutyric acid 153548-49-3 164667-45-2, 2-Formyl-4H-furo[3,2-b]pyrrole-5-carboxylic acid 186431-46-9 186432-05-3 238749-50-3, 2-Bromo-4H-thieno[3,2-b]pyrrole-5-carboxylic acid ethyl ester 519188-80-8
RL: RCT (Reactant); RACT (Reactant or reagent)
(preparation of fused pyrrolylcarboxamides as glycogen phosphorylase inhibitors)

IT 65782-04-9P, 5-Chloro-4-methylthiophene-2-carboxaldehyde 238749-54-7P, 2-Methyl-4H-thieno[3,2-b]pyrrole-5-carboxylic acid ethyl ester 332098-79-0P 332098-81-4P 332098-83-6P, 2-Bromo-6H-thieno[2,3-b]pyrrole-5-carboxylic acid 332098-85-8P, 2-Methyl-6H-thieno[2,3-b]pyrrole-5-carboxylic acid ethyl ester 332098-87-0P, 2-Methyl-6H-thieno[2,3-b]pyrrole-5-carboxylic acid 332098-89-2P 332098-91-6P 332098-93-8P 332098-95-0P 332098-97-2P 332098-99-4P 332099-01-1P, 2-Chloro-6H-thieno[2,3-b]pyrrole-5-carboxylic acid ethyl ester 332099-03-3P, 2-Chloro-6H-thieno[2,3-b]pyrrole-5-carboxylic acid 332099-05-5P, 2,4-Dichloro-6H-thieno[2,3-b]pyrrole-5-carboxylic acid ethyl ester 332099-07-7P, 2,4-Dichloro-6H-thieno[2,3-b]pyrrole-5-carboxylic acid 332099-09-9P, 2-Bromo-4H-thieno[3,2-b]pyrrole-5-carboxylic acid 332099-11-3P, 2-Bromo-4H-furo[3,2-b]pyrrole-5-carboxylic acid 332099-14-6P, 2-Methyl-4H-thieno[3,2-b]pyrrole-5-carboxylic acid 332099-16-8P, 2-Cyano-6H-thieno[2,3-b]pyrrole-5-carboxylic acid 332099-18-0P 332099-20-4P 332099-22-6P, 2-Fluoro-4H-thieno[3,2-b]pyrrole-5-carboxylic acid ethyl ester 332099-24-8P, 2-Fluoro-4H-thieno[3,2-b]pyrrole-5-carboxylic acid 332099-26-0P,

2-Cyano-4H-furo[3,2-b]pyrrole-5-carboxylic acid 332099-28-2P, 2-Chloro-4H-furo[3,2-b]pyrrole-5-carboxylic acid ethyl ester 332099-29-3P, 2-Chloro-4H-furo[3,2-b]pyrrole-5-carboxylic acid 332099-31-7P, 3-Chloro-4H-thieno[3,2-b]pyrrole-5-carboxylic acid ethyl ester 332099-33-9P, 3-Chloro-4H-thieno[3,2-b]pyrrole-5-carboxylic acid 332099-35-1P, 3-Bromo-4H-thieno[3,2-b]pyrrole-5-carboxylic acid ethyl ester 332099-36-2P, 3-Bromo-4H-thieno[3,2-b]pyrrole-5-carboxylic acid 332099-38-4P, 2-Chloro-4H-thieno[3,2-b]pyrrole-5-carboxylic acid ethyl ester 332099-40-8P, 2-Chloro-4H-thieno[3,2-b]pyrrole-5-carboxylic acid 332099-42-0P, 3-Methyl-4H-thieno[3,2-b]pyrrole-5-carboxylic acid ethyl ester 332099-44-2P, 3-Methyl-4H-thieno[3,2-b]pyrrole-5-carboxylic acid 332099-46-4P, 2-Formyl-4H-thieno[3,2-b]pyrrole-5-carboxylic acid 332099-48-6P, 2-Cyano-4H-thieno[3,2-b]pyrrole-5-carboxylic acid 332099-50-0P, 3-Bromo-4H-furo[3,2-b]pyrrole-5-carboxylic acid ethyl ester 332099-52-2P, 3-Bromo-4H-furo[3,2-b]pyrrole-5-carboxylic acid 332099-54-4P 332099-55-5P 332099-56-6P, 2-Chloro-3-methyl-4H-thieno[3,2-b]pyrrole-5-carboxylic acid ethyl ester 332099-58-8P, 2-Chloro-3-methyl-4H-thieno[3,2-b]pyrrole-5-carboxylic acid 332099-60-2P 332099-62-4P
RL: RCT (Reactant); SPN (Synthetic preparation); PREP (Preparation); RACT (Reactant or reagent)
(preparation of fused pyrrolylcarboxamides as glycogen phosphorylase inhibitors)
RE.CNT 3 THERE ARE 3 CITED REFERENCES AVAILABLE FOR THIS RECORD
RE
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(2) Hitzel, V; US 4325963 A 1982 CAPLUS
(3) Pfizer; EP 0846464 A 1998 CAPLUS

L34 ANSWER 3 OF 9 CAPLUS COPYRIGHT 2007 ACS on STN
ACCESSION NUMBER: 2003:570833 CAPLUS <<LOGINID::20070124>>
DOCUMENT NUMBER: 139:111682
TITLE: Combined use of a GLP-1 compound and a modulator of diabetic late complications
INVENTOR(S): Knudsen, Lotte Bjerre; Selmer, Johan
PATENT ASSIGNEE(S): Novo Nordisk A/S, Den.
SOURCE: PCT Int. Appl., 22 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2003059372	A2	20030724	WO 2002-DK888	20021220 <--
WO 2003059372	A3	20040325		

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, UZ, VC, VN, YU, ZA, ZM, ZW
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG

AU 2002351753	A1	20030730	AU 2002-351753	20021220 <--
EP 1461070	A2	20040929	EP 2002-787467	20021220

R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR, BG, CZ, EE, SK

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US 2003144206	A1	20030731	US 2002-328282	20021223 <--

PRIORITY APPLN. INFO.: DK 2001-1969 A 20011229
DK 2002-760 A 20020517
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US 2002-350087P P 20020117
WO 2002-DK888 W 20021220
AN 2003:570833 CAPLUS <<LOGINID::20070124>>
DN 139:111682
ED Entered STN: 25 Jul 2003
TI Combined use of a GLP-1 compound and a modulator of diabetic late complications
IN Knudsen, Lotte Bjerre; Selmer, Johan
PA Novo Nordisk A/S, Den.
SO PCT Int. Appl., 22 pp.
CODEN: PIXXD2
DT Patent
LA English
IC ICM A61K038-00
CC 1-10 (Pharmacology)
Section cross-reference(s): 2, 63
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI WO 2003059372	A2	20030724	WO 2002-DK888	20021220 <--
WO 2003059372	A3	20040325		

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RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG

AU 2002351753	A1	20030730	AU 2002-351753	20021220 <--
EP 1461070	A2	20040929	EP 2002-787467	20021220

R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR, BG, CZ, EE, SK

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PRAI DK 2001-1969 A 20011229
DK 2002-760 A 20020517
DK 2001-969 A 20011229
US 2002-350087P P 20020117
WO 2002-DK888 W 20021220
CLASS

PATENT NO.	CLASS	PATENT FAMILY CLASSIFICATION CODES
WO 2003059372	ICM	A61K038-00
	IPCI	A61K0038-00 [ICM,7]
	IPCR	A61K0045-00 [I,C*]; A61K0045-00 [I,A]; A61K0031-138 [I,C*]; A61K0031-138 [I,A]; A61K0031-165 [I,C*]; A61K0031-165 [I,A]; A61K0031-167 [I,C*]; A61K0031-167 [I,A]; A61K0031-21 [I,C*]; A61K0031-216 [I,A]; A61K0031-35 [I,C*]; A61K0031-35 [I,A]; A61K0031-401 [I,C*]; A61K0031-401 [I,A]; A61K0031-403 [I,C*]; A61K0031-403 [I,A]; A61K0031-404 [I,A]; A61K0031-407 [I,C*]; A61K0031-407 [I,A]; A61K0031-4164 [I,C*]; A61K0031-4166 [I,A]; A61K0031-4184 [I,A]; A61K0031-4188 [I,A]; A61K0031-4196 [I,C*]; A61K0031-4196 [I,A]; A61K0031-472 [I,C*]; A61K0031-472 [I,A]; A61K0031-5375 [I,C*]; A61K0031-5377 [I,A]; A61K0031-55 [I,C*]; A61K0031-55 [I,A]; A61K0038-00 [I,C*]; A61K0038-00 [I,A]; A61K0038-26 [I,C*]; A61K0038-26 [I,A]; A61P0003-00 [I,C*]; A61P0003-10 [I,A]; A61P0009-00 [I,C*]; A61P0009-10 [I,A]; A61P0009-12 [I,A]; A61P0013-00 [I,C*]; A61P0013-12 [I,A]; A61P0025-00 [I,C*]; A61P0025-02 [I,A]; A61P0027-00 [I,C*]; A61P0027-02 [I,A]; A61P0043-00 [I,C*]; A61P0043-00 [I,A]
AU 2002351753	IPCI	A61K0038-00 [ICM,7]
	IPCR	A61K0045-00 [I,C*]; A61K0045-00 [I,A]; A61K0031-138 [I,C*]; A61K0031-138 [I,A]; A61K0031-165 [I,C*]; A61K0031-165 [I,A]; A61K0031-167 [I,C*]; A61K0031-167 [I,A]; A61K0031-21 [I,C*]; A61K0031-216 [I,A]; A61K0031-35 [I,C*]; A61K0031-35 [I,A]; A61K0031-401 [I,C*]; A61K0031-401 [I,A]; A61K0031-403 [I,C*]; A61K0031-403 [I,A]; A61K0031-404 [I,A]; A61K0031-407 [I,C*]; A61K0031-407 [I,A]; A61K0031-4164 [I,C*]; A61K0031-4166 [I,A]; A61K0031-4184 [I,A]; A61K0031-4188 [I,A]; A61K0031-4196 [I,C*]; A61K0031-4196 [I,A]; A61K0031-472 [I,C*]; A61K0031-472 [I,A]; A61K0031-5375 [I,C*]; A61K0031-5377 [I,A]; A61K0031-55 [I,C*]; A61K0031-55 [I,A]; A61K0038-00 [I,C*]; A61K0038-00 [I,A]; A61K0038-26 [I,C*]; A61K0038-26 [I,A]; A61P0003-00 [I,C*]; A61P0003-10 [I,A]; A61P0009-00 [I,C*]; A61P0009-10 [I,A]; A61P0009-12 [I,A]; A61P0013-00 [I,C*]; A61P0013-12 [I,A]; A61P0025-00 [I,C*]; A61P0025-02 [I,A]; A61P0027-00 [I,C*]; A61P0027-02 [I,A]; A61P0043-00 [I,C*]; A61P0043-00 [I,A]
EP 1461070	IPCI	A61K0038-26 [ICM,7]; A61K0031-35 [ICS,7]; A61P0003-10 [ICS,7]; A61P0003-00 [ICS,7,C*]
	IPCR	A61K0045-00 [I,C*]; A61K0045-00 [I,A]; A61K0031-138 [I,C*]; A61K0031-138 [I,A]; A61K0031-165 [I,C*]; A61K0031-165 [I,A]; A61K0031-167 [I,C*]; A61K0031-167 [I,A]; A61K0031-21 [I,C*]; A61K0031-216 [I,A]; A61K0031-35 [I,C*]; A61K0031-35 [I,A]; A61K0031-401 [I,C*]; A61K0031-401 [I,A]; A61K0031-403 [I,C*]; A61K0031-403 [I,A]; A61K0031-404 [I,A]; A61K0031-407 [I,C*]; A61K0031-407 [I,A]; A61K0031-4164 [I,C*]; A61K0031-4166 [I,A]; A61K0031-4184 [I,A]; A61K0031-4188 [I,A]; A61K0031-4196 [I,C*]; A61K0031-4196 [I,A]; A61K0031-472 [I,C*]; A61K0031-472 [I,A]; A61K0031-5375 [I,C*]; A61K0031-5377 [I,A]; A61K0031-55 [I,C*]; A61K0031-55 [I,A]; A61K0038-00 [I,C*]; A61K0038-00 [I,A]; A61K0038-26 [I,C*]; A61K0038-26 [I,A]; A61P0003-00 [I,C*]; A61P0003-10 [I,A]; A61P0009-00 [I,C*]; A61P0009-10 [I,A]; A61P0009-12 [I,A]; A61P0013-00 [I,C*]; A61P0013-12 [I,A]; A61P0025-00 [I,C*]; A61P0025-02 [I,A]; A61P0027-00 [I,C*]; A61P0027-02 [I,A]; A61P0043-00 [I,C*]; A61P0043-00 [I,A]
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A61K0031-472 [ICS,7]; A61K0031-5377 [ICS,7]; A61K0031-5375 [ICS,7,C*]; A61K0031-55 [ICS,7]; A61K0045-00 [ICS,7]; A61P0003-10 [ICS,7]; A61P0003-00 [ICS,7,C*]; A61P0009-10 [ICS,7]; A61P0009-12 [ICS,7]; A61P0009-00 [ICS,7,C*]; A61P0013-12 [ICS,7]; A61P0013-00 [ICS,7,C*]; A61P0025-00 [ICS,7]; A61P0025-02 [ICS,7]; A61P0027-02 [ICS,7]; A61P0027-00 [ICS,7,C*]; A61P0043-00 [ICS,7]

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FTERM 4C084/AA02; 4C084/AA03; 4C084/AA19; 4C084/BA19; 4C084/CA18; 4C084/CA59; 4C084/MA02; 4C084/MA52; 4C084/MA55; 4C084/MA56; 4C084/MA59; 4C084/MA63; 4C084/NA05; 4C084/NA06; 4C084/ZA021; 4C084/ZA262; 4C084/ZA331; 4C084/ZA361; 4C084/ZA421; 4C084/ZA422; 4C084/ZA811; 4C084/ZC202; 4C084/ZC351; 4C084/ZC422; 4C086/AA01; 4C086/AA02; 4C086/BC07; 4C086/BC10; 4C086/BC13; 4C086/BC30; 4C086/BC32; 4C086/BC38; 4C086/BC62; 4C086/BC85; 4C086/CB22; 4C086/CB27; 4C086/GA07; 4C086/GA10; 4C086/GA12; 4C086/MA02; 4C086/MA04; 4C086/MA07; 4C086/MA52; 4C086/MA55; 4C086/NA05; 4C086/NA06; 4C086/ZA02; 4C086/ZA26; 4C086/ZA33; 4C086/ZA36; 4C086/ZA42; 4C086/ZA81; 4C086/ZC20; 4C086/ZC35; 4C086/ZC42; 4C206/AA01; 4C206/AA02; 4C206/FA18; 4C206/FA19; 4C206/FA21; 4C206/GA01; 4C206/GA31; 4C206/KA01; 4C206/MA02; 4C206/MA04; 4C206/MA11; 4C206/MA72; 4C206/MA75; 4C206/NA05; 4C206/NA06; 4C206/ZA02; 4C206/ZA26; 4C206/ZA33; 4C206/ZA36; 4C206/ZA42; 4C206/ZA81; 4C206/ZC20; 4C206/ZC35; 4C206/ZC42

US 2003144206 IPCI A61K0038-26 [ICM,7]; A61K0031-401 [ICS,7]
IPCR A61K0031-401 [I,C*]; A61K0031-401 [I,A]; A61K0038-26 [I,C*]; A61K0038-26 [I,A]
NCL 514/012.000; 514/423.000

AB Methods and uses for treatment of diabetic late complications comprising administration of a GLP-1 compound and a modulator of diabetic complications.

ST GLP1 diabetes late complication therapy; glucagon like peptide I analog fragment antidiabetic

IT Angiotensin receptor antagonists
Antihypertensives
Human
Hypertension
Protein sequences
b-Adrenoceptor antagonists
b1-Adrenoceptor antagonists
(combined use of a GLP-1 compound and a modulator of diabetic late complications)

IT Kidney, disease
(diabetic nephropathy; combined use of a GLP-1 compound and a modulator of diabetic late complications)

IT Nerve, disease
(diabetic neuropathy; combined use of a GLP-1 compound and a modulator of diabetic late complications)

IT Eye, disease
(diabetic retinopathy; combined use of a GLP-1 compound and a modulator of diabetic late complications)

IT Gene, animal
RL: PAC (Pharmacological activity); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(glp-1; combined use of a GLP-1 compound and a modulator of diabetic late complications)

IT Diabetes mellitus
(non-insulin-dependent; combined use of a GLP-1 compound and a modulator of diabetic late complications)

IT Antidiabetic agents
Drug delivery systems
(oral; combined use of a GLP-1 compound and a modulator of diabetic late complications)

IT Drug delivery systems
(parenterals; combined use of a GLP-1 compound and a modulator of diabetic late complications)

IT 496765-91-4
RL: PAC (Pharmacological activity); PRP (Properties); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(amino acid sequence; combined use of a GLP-1 compound and a modulator of diabetic late complications)

IT 525-66-6, Propranolol 13523-86-9, Pindolol 26839-75-8, Timolol 29122-68-7, Atenolol 37517-30-9, Acebutolol 42200-33-9, Nadolol 51384-51-1, Metoprolol 62571-86-2, Captopril 75847-73-3, Enalapril 76547-98-3, Lisinopril 81147-92-4, Esmolol 83647-97-6, Spirapril 85441-61-8, Quinapril 86541-75-5, Benazepril 87333-19-5, Ramipril 87679-37-6, Trandolapril 89371-37-9, Imidapril 89750-14-1D, GLP-1, analogs or fragments 98048-97-6, Fosinopril 107133-36-8, Perindopril erbumine 114798-26-4, Losartan 135038-57-2, Alatriopril 136087-85-9, Fidarestat 137862-53-4, Valsartan 138402-11-6, Irbesartan 141732-76-5, Exendin-4 141732-76-5D, Exendin-4, derivs. 169939-94-0, Ly 333531
RL: PAC (Pharmacological activity); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(combined use of a GLP-1 compound and a modulator of diabetic late complications)

IT 9015-82-1, Angiotensin-converting enzyme 9028-31-3, Aldose reductase 141436-78-4, Protein kinase C
RL: PAC (Pharmacological activity); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(inhibitors; combined use of a GLP-1 compound and a modulator of diabetic late complications)

L34 ANSWER 4 OF 9 CAPLUS COPYRIGHT 2007 ACS on STN

ACCESSION NUMBER: 2003:320036 CAPLUS <<LOGINID::20070124>>
DOCUMENT NUMBER: 138:338498
TITLE: Preparation of human glucagon-like-peptide-1 mimics and their use in the treatment of diabetes and related conditions
INVENTOR(S): Natarajan, Sesha I.; Bastos, Margarita M.; Bernatowicz, Michael S.; Mapelli, Claudio; Lee, Ving; Ewing, William R.
PATENT ASSIGNEE(S): Bristol-Myers Squibb Company, USA
SOURCE: PCT Int. Appl., 153 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 2
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2003033671	A2	20030424	WO 2002-US33386	20021018 <--
WO 2003033671	A3	20051229		

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG

CA 2463908	A1	20030424	CA 2002-2463908	20021018 <--
JP 2005514337	T	20050519	JP 2003-536401	20021018
CN 1630709	A	20050622	CN 2002-820558	20021018
EP 1572892	A2	20050914	EP 2002-782185	20021018

R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR, BG, CZ, EE, SK

BR 2002013377	A	20060523	BR 2002-13377	20021018
NO 2004001203	A	20040610	NO 2004-1203	20040323
ZA 2004002846	A	20050816	ZA 2004-2846	20040415

PRIORITY APPLN. INFO.: US 2001-342015P P 20011018
WO 2002-US33386 W 20021018
OTHER SOURCE(S): MARPAT 138:338498
AN 2003:320036 CAPLUS <<LOGINID::20070124>>
DN 138:338498
ED Entered STN: 25 Apr 2003
TI Preparation of human glucagon-like-peptide-1 mimics and their use in the treatment of diabetes and related conditions
IN Natarajan, Sesha I.; Bastos, Margarita M.; Bernatowicz, Michael S.; Mapelli, Claudio; Lee, Ving; Ewing, William R.
PA Bristol-Myers Squibb Company, USA
SO PCT Int. Appl., 153 pp.
CODEN: PIXXD2
DT Patent
LA English
IC ICM C12N
CC 34-3 (Amino Acids, Peptides, and Proteins)
Section cross-reference(s): 1, 63
FAN.CNT 2

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI WO 2003033671	A2	20030424	WO 2002-US33386	20021018 <--
WO 2003033671	A3	20051229		

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NO 2004001203	A	20040610	NO 2004-1203	20040323
ZA 2004002846	A	20050816	ZA 2004-2846	20040415
PRAI US 2001-342015P	P	20011018		
WO 2002-US33386	W	20021018		

CLASS

PATENT NO.	CLASS	PATENT FAMILY CLASSIFICATION CODES
WO 2003033671	ICM	C12N
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CA 2463908	IPCI	C07K0014-605 [ICM,7]; C07K0014-435 [ICM,7,C*]; C07K0007-06 [ICS,7]; C07K0007-00 [ICS,7,C*]; A61K0038-08 [ICS,7]; A61K0038-26 [ICS,7]
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BR 2002013377	IPCI	A61K0038-00 [ICS,7]; A61K0038-08 [ICS,7]; C07K0002-00 [ICS,7]
	IPCR	A61K0038-00 [N,C*]; C07K0014-435 [I,C*]; A61K0038-00 [N,A]; C07K0014-605 [I,A]
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NO 2004001203	IPCI	C07K0014-605 [ICM,7]; C07K0014-435 [ICM,7,C*]; C07K0004-00 [ICS,7]; A61K0038-26 [ICS,7]; A61K0038-08 [ICS,7]; A61K0038-10 [ICS,7]
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ZA 2004002846	IPCR	A61K0038-00 [N,C*]; C07K0014-435 [I,C*]; A61K0038-00 [N,A]; C07K0014-605 [I,A]
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JP 2005514337	IPCI	C07K0007-06 [ICM,7]; A61K0038-00 [ICS,7]; A61P0003-04 [ICS,7]; A61P0003-06 [ICS,7]; A61P0003-10 [ICS,7]; A61P0003-00 [ICS,7,C*]; A61P0005-50 [ICS,7]; A61P0005-00 [ICS,7,C*]; A61P0009-10 [ICS,7]; A61P0009-12 [ICS,7]; A61P0009-00 [ICS,7,C*]; A61P0013-12 [ICS,7]; A61P0013-00 [ICS,7,C*]; A61P0017-02 [ICS,7]; A61P0017-00 [ICS,7,C*]; A61P0025-00 [ICS,7]; A61P0027-02 [ICS,7]; A61P0027-00 [ICS,7,C*]; A61P0043-00 [ICS,7]; C07K0007-08 [ICS,7]; C07K0007-00 [ICS,7,C*]; C07K0014-00 [ICS,7]; A61K0038-26 [ICS,7]
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CN 1630709	IPCI	C12N0001-00 [ICM,7]
	IPCR	A61K0038-00 [I,C*]; A61K0038-00 [I,A]; A61K0038-26 [I,C*]; A61K0038-26 [I,A]; A61P0003-00 [I,C*]; A61P0003-04 [I,A]; A61P0003-06 [I,A]; A61P0003-10 [I,A]; A61P0005-00 [I,C*]; A61P0005-50 [I,A]; A61P0009-00 [I,C*]; A61P0009-10 [I,A]; A61P0009-12 [I,A]; A61P0013-00 [I,C*]; A61P0013-12 [I,A]; A61P0017-00 [I,C*]; A61P0017-02 [I,A]; A61P0025-00 [I,C*]; A61P0025-00 [I,A]; A61P0027-00 [I,C*]; A61P0027-02 [I,A]; A61P0043-00 [I,C*]; A61P0043-00 [I,A]; C07K0007-00 [I,C*]; C07K0007-06 [I,A]; C07K0007-08 [I,A]; C07K0014-00 [I,C*]; C07K0014-00 [I,A]; C07K0014-435 [I,C*]; C07K0014-605 [I,A]
EP 1572892	IPCI	C12N0001-00 [ICM,7]
	IPCR	A61K0038-00 [I,C*]; A61K0038-00 [I,A]; A61K0038-08 [I,C]; A61K0038-08 [I,A]; A61K0038-26 [I,C*]; A61K0038-26 [I,A]; A61P0003-00 [I,C*]; A61P0003-04 [I,A]; A61P0003-06 [I,A]; A61P0003-10 [I,A]; A61P0005-00 [I,C*]; A61P0005-50 [I,A]; A61P0009-00 [I,C*]; A61P0009-10 [I,A]; A61P0009-12 [I,A]; A61P0013-00 [I,C*]; A61P0013-12 [I,A]; A61P0017-00 [I,C*]; A61P0017-02 [I,A]; A61P0025-00 [I,C*]; A61P0025-00 [I,A]; A61P0027-00 [I,C*]; A61P0027-02 [I,A]; A61P0043-00 [I,C*]; A61P0043-00 [I,A]; C07K0002-00 [I,C]; C07K0002-00 [I,A]; C07K0007-00 [I,C*]; C07K0007-08 [I,A]; C07K0014-00 [I,C*]; C07K0014-00 [I,A]; C07K0014-435 [I,C*]; C07K0014-605 [I,A]

OS MARPAT 138:338498

AB The invention provides novel human glucagon-like peptide-1 (GLP-1) peptide mimics A-Xaa1-Xaa2-Xaa3-Xaa4-Xaa5-Xaa6-Xaa7-Xaa8-Xaa9-Y-Z-B [Xaa1-Xaa9 are naturally or non-naturally occurring amino acid residues; Y and Z are amino acid residues which may be substituted; A and B are optionally present; A is H, an amino acid or peptide containing .apprx. 1-15 amino acid residues, an R group [H, (cyclo)alkyl, cycloalkylalkyl, heterocyclyl, heterocycloalkyl, (hetero)aryl, arylalkyl, aryloxyalkyl, heteroarylalkyl, or heteroaryloxyalkyl], an RCO (amide) group, a carbamate group, a urea, a sulfonamido, or an aminosulfonyl group; B is OH, alkoxy, etc., an amino or amino acid residue, or a peptide containing from 1-15 amino acid residues, terminating at the C-terminus as a carboxamide, ester, carboxyl, or an amino alc.] that mimic the biol. activity of the native GLP-1 peptide and thus are useful for the treatment or prevention of diseases or disorders associated with GLP activity. These chemical-modified peptides stimulate insulin secretion in type II diabetics and produce other beneficial insulinotropic responses, while exhibiting increased stability to proteolytic cleavage making them ideal therapeutic candidates for oral or parenteral administration. A method of preparing the polypeptides comprises replacing the message sequence of the polypeptide with a variant message sequence capable of inducing receptor mediated signal transduction. An example is claimed peptide H-AEGTFTSD-Bip(2-Et)-Bip(2-Me)-NH2 (Bip = biphenylalanine residue).

ST glucagon like peptide mimic prepn treatment diabetes

IT Antiarteriosclerotics
(antiatherosclerotics; preparation of human glucagon-like-peptide-1 mimics for use in treatment of diabetes and related conditions)

IT Kidney, disease
(diabetic nephropathy; preparation of human glucagon-like-peptide-1 mimics for use in treatment of diabetes and related conditions)

IT Nerve, disease
(diabetic neuropathy; preparation of human glucagon-like-peptide-1 mimics for use in treatment of diabetes and related conditions)

IT Eye, disease
(diabetic retinopathy; preparation of human glucagon-like-peptide-1 mimics for use in treatment of diabetes and related conditions)

IT Metabolic disorders
(metabolic syndrome X; preparation of human glucagon-like-peptide-1 mimics for use in treatment of diabetes and related conditions)

IT Antidiabetic agents
Antihypertensives
Antiobesity agents
Atherosclerosis
Diabetes mellitus
Human
Hyperglycemia
Hypertension
Hypertriglyceridemia
Hypolipemic agents
Obesity
Wound healing
(preparation of human glucagon-like-peptide-1 mimics for use in treatment of diabetes and related conditions)

IT Hyperlipidemia
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(preparation of human glucagon-like-peptide-1 mimics for use in treatment of diabetes and related conditions)

IT Peptides, preparation
RL: PAC (Pharmacological activity); SPN (Synthetic preparation); THU

(Therapeutic use); BIOL (Biological study); PREP (Preparation); USES (Uses)
(preparation of human glucagon-like-peptide-1 mimics for use in treatment of diabetes and related conditions)

IT 89750-14-1DP, Glucagon-like peptide I, mimics 516514-32-2P
516514-38-8P 516514-43-5P 516514-47-9P 516514-52-6P 516514-55-9P
516514-58-2P 516514-61-7P 516514-64-0P 516514-68-4P 516514-72-0P
516514-75-3P 516514-78-6P 516514-81-1P 516514-84-4P 516514-87-7P
516514-91-3P 516514-95-7P 516514-99-1P 516515-03-0P 516515-06-3P
516515-09-6P 516515-14-3P 516515-18-7P 516515-22-3P 516515-26-7P
516515-30-3P 516515-34-7P 516515-38-1P 516515-42-7P 516515-46-1P
516515-50-7P 516515-55-2P 516515-59-6P 516515-63-2P 516515-68-7P
516515-72-3P 516515-76-7P 516515-80-3P 516515-84-7P 516515-88-1P
516515-92-7P 516515-96-1P 516516-01-1P 516516-06-6P 516516-10-2P
516516-14-6P 516516-18-0P 516516-22-6P 516516-26-0P 516516-31-7P
516516-35-1P 516516-39-5P 516516-44-2P 516516-50-0P 516516-55-5P
516516-60-2P 516516-64-6P 516516-68-0P 516516-72-6P 516516-76-0P
516516-80-6P 516516-85-1P 516516-87-3P 516516-91-9P 516516-95-3P
516516-98-6P 516517-02-5P 516517-06-9P 516517-10-5P 516517-14-9P
516517-17-2P 516517-22-9P 516517-26-3P 516517-30-9P 516517-33-2P
516517-37-6P 516517-41-2P 516517-45-6P 516517-50-3P 516517-54-7P
516517-59-2P 516517-63-8P 516517-67-2P 516517-71-8P 516517-75-2P
516517-79-6P 516517-82-1P 516517-85-4P 516517-88-7P 516517-91-2P
516517-96-7P 516518-00-6P 516518-04-0P 516518-08-4P 516518-11-9P
516518-15-3P 516518-19-7P 516518-22-2P 516518-26-6P 516518-30-2P
516518-33-5P 516518-35-7P 516518-39-1P 516518-42-6P 516518-46-0P
516518-48-2P 516518-51-7P 516518-54-0P 516518-57-3P 516518-59-5P
516518-61-9P 516518-64-2P 516518-66-4P 516518-69-7P 516518-73-3P
516518-78-8P 516518-83-5P 516518-88-0P 516518-92-6P 516518-96-0P
516519-00-9P 516519-04-3P 516519-09-8P 516519-12-3P 516519-15-6P
516519-18-9P 516519-21-4P 516519-24-7P 516519-27-0P 516519-32-7P
516519-37-2P 516519-40-7P 516519-45-2P 516519-50-9P 516519-54-3P
516519-59-8P 516519-63-4P 516519-67-8P 516519-72-5P 516519-77-0P
516519-82-7P 516519-87-2P 516519-91-8P 516519-95-2P 516519-99-6P
516520-03-9P 516520-09-5P 516520-13-1P 516520-17-5P 516520-22-2P
516520-26-6P 516520-29-9P 516520-33-5P 516520-36-8P 516520-39-1P
516520-42-6P 516520-45-9P 516520-47-1P 516520-49-3P 516520-52-8P
516520-54-0P 516520-55-1P 516520-57-3P 516520-59-5P 516520-61-9P
516520-63-1P 516520-66-4P 516520-68-6P 516520-70-0P 516520-72-2P
516520-74-4P 516520-75-5P 516520-77-7P 516520-79-9P 516520-81-3P
516520-82-4P 516520-84-6P 516520-86-8P 516520-87-9P 516520-89-1P
516520-91-5P 516520-93-7P 516520-95-9P 516520-97-1P 516520-99-3P
516521-01-0P 516521-03-2P 516521-05-4P 516521-07-6P 516521-08-7P
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516521-16-7P 516521-18-9P 516521-19-0P 516521-21-4P 516521-22-5P
516521-23-6P 516521-24-7P 516521-25-8P 516521-26-9P 516521-27-0P
516521-28-1P 516521-29-2P 516521-30-5P 516521-31-6P 516521-32-7P
516521-33-8P 516521-34-9P 516521-35-0P 516521-36-1P 516521-37-2P
516521-38-3P 516521-39-4P 516521-40-7P 516521-41-8P 516521-42-9P
516521-43-0P 516521-44-1P 516521-45-2P 516521-53-2P 516521-54-3P
516521-55-4P
RL: PAC (Pharmacological activity); SPN (Synthetic preparation); THU (Therapeutic use); BIOL (Biological study); PREP (Preparation); USES (Uses)
(preparation of human glucagon-like-peptide-1 mimics for use in treatment of diabetes and related conditions)

IT 150-46-9, Triethyl borate 1973-22-4, 1 Bromo 2 ethylbenzene 4326-36-7
16419-60-6, o Tolylboronic acid 93267-04-0 516521-49-6
RL: RCT (Reactant); RACT (Reactant or reagent)
(preparation of human glucagon-like-peptide-1 mimics for use in treatment of diabetes and related conditions)

IT 90002-36-1P, 2 Ethylphenylboronic acid 112766-18-4P 516521-46-3P
516521-47-4P 516521-48-5P 516521-50-9P 516521-51-0P
RL: RCT (Reactant); SPN (Synthetic preparation); PREP (Preparation); RACT (Reactant or reagent)
(preparation of human glucagon-like-peptide-1 mimics for use in treatment of diabetes and related conditions)

IT 51-64-9, Dexamphetamine 94-20-2, Chloropropamide 122-09-8, Phentermine 637-07-0, Clofibrate 657-24-9, Metformin 9004-10-8, Insulin, biological studies 10238-21-8, Glyburide 14838-15-4, Phenylpropanolamine 21187-98-4, Gliclazide 22232-71-9, Mazindol 25812-30-0, Gemfibrozil 29094-61-9, Glipizide 49562-28-9, Fenofibrate 56180-94-0, Acarbose 72432-03-2, Miglitol 75330-75-5, Lovastatin 79902-63-9, Simvastatin 81093-37-0, Pravastatin 93479-97-1, Glimepiride 93957-54-1, Fluvastatin 96829-58-2, Orlistat 97240-79-4, Topiramate 97322-87-7, Troglitazone 105816-04-4, Nateglinide 106650-56-0, Sibutramine 111025-46-8, Pioglitazone 122320-73-4, Rosiglitazone 134523-00-5, Atorvastatin 135062-02-1, Repaglinide 141750-63-2, Nisvastatin 141758-74-9, AC 2993 144288-97-1, TS-962 145599-86-6, Cerivastatin 152755-31-2, LY295427 159183-92-3, L750355 161600-01-7, Isaglitazone 166518-60-1, Avasimibe 170861-63-9, JTT-501 176435-10-2, LY315902 178759-95-0, MD 700 196808-45-4 199113-98-9 199914-96-0, YM-440 213252-19-8, KRP297 244081-42-3, AJ9677 258345-41-4, GW-409544 262352-17-0, CP 529414 282526-98-1, ATL-962 287714-41-4 335149-08-1, L895645 335149-14-9, R-119702 335149-15-0, KAD1129 335149-17-2, ARHO39242 335149-23-0, NVPDPP-728A 335149-25-2,
CP331648 430433-17-3, Glipyride 444069-80-1, Axokine
RL: THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(preparation of human glucagon-like-peptide-1 mimics for use in treatment of diabetes and related conditions)

L34 ANSWER 5 OF 9 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on STN
ACCESSION NUMBER: 2003:390202 BIOSIS <<LOGINID::20070124>>
DOCUMENT NUMBER: PREV200300390202
TITLE: The glucagon-like peptides: A double-edged therapeutic sword?.
AUTHOR(S): Perry, TracyAnn [Reprint Author]; Greig, Nigel H.
CORPORATE SOURCE: Section of Drug Design and Development, Laboratory of Neurosciences, Gerontology Research Center, National Institute on Aging, National Institutes of Health, 5600 Nathan Shock Drive, Baltimore, MD, 21224, USA
perryt@grc.nia.nih.gov
SOURCE: Trends in Pharmacological Sciences, (July 2003) Vol. 24, No. 7, pp. 377-383. print.
ISSN: 0165-6147 (ISSN print).
DOCUMENT TYPE: Article
General Review; (Literature Review)
LANGUAGE: English
ENTRY DATE: Entered STN: 27 Aug 2003
Last Updated on STN: 27 Aug 2003
AN 2003:390202 BIOSIS <<LOGINID::20070124>>
DN PREV200300390202
TI The glucagon-like peptides: A double-edged therapeutic sword?.
AU Perry, TracyAnn [Reprint Author]; Greig, Nigel H.
CS Section of Drug Design and Development, Laboratory of Neurosciences, Gerontology Research Center, National Institute on Aging, National Institutes of Health, 5600 Nathan Shock Drive, Baltimore, MD, 21224, USA
perryt@grc.nia.nih.gov
SO Trends in Pharmacological Sciences, (July 2003) Vol. 24, No. 7, pp. 377-383. print.
ISSN: 0165-6147 (ISSN print).
DT Article
General Review; (Literature Review)
LA English
ED Entered STN: 27 Aug 2003
Last Updated on STN: 27 Aug 2003
AB Glucagon-like peptide-1(7-36)-amide (GLP-1) is an endogenous peptide that is secreted from the gut in response to the presence of food. Recent studies have established that GLP-1 and its longer-acting analog exendin-4 have multiple synergistic effects on glucose-dependent, insulin secretion pathways of the pancreatic beta-cell and on plasticity in neuronal cells. Recent interest has focused on the development of these peptides as a novel therapeutic strategy for non-insulin-dependent (type 2) diabetes mellitus and associated neuropathy. This is with a view to developing lead compounds, based on neurotrophic action, for central and peripheral degenerative disorders such as stroke and Alzheimer's disease in addition to the peripheral neuropathy associated with type 2 diabetes mellitus. Here, we address recent advances in the biological action of GLP-1 and its related analogs.
CC Cytology - Animal 02506
Biochemistry studies - Proteins, peptides and amino acids 10064
Biochemistry studies - Carbohydrates 10068
Pathology - Therapy 12512
Metabolism - Metabolic disorders 13020
Cardiovascular system - Blood vessel pathology 14508
Endocrine - General 17002
Endocrine - Pancreas 17008
Endocrine - Neuroendocrinology 17020
Nervous system - Physiology and biochemistry 20504
Nervous system - Pathology 20506
Pharmacology - General 22002
IT Major Concepts
Endocrine System (Chemical Coordination and Homeostasis); Nervous System (Neural Coordination); Pharmacology
IT Parts, Structures, & Systems of Organisms
beta cells: endocrine system; neuronal cells: nervous system
IT Diseases
Alzheimer's disease: behavioral and mental disorders, nervous system disease
Alzheimer Disease (MeSH)
IT Diseases
diabetic neuropathy: endocrine disease/pancreas, metabolic disease, nervous system disease
Diabetic Nephropathies (MeSH)
IT Diseases
stroke: nervous system disease, vascular disease
Cerebrovascular Disorders (MeSH)
IT Diseases
type 2 diabetes mellitus: endocrine disease/pancreas, metabolic disease
Diabetes Mellitus, Non-Insulin-Dependent (MeSH)
IT Chemicals & Biochemicals
glucagon-like peptide-1(7-36)-amide; glucose; insulin
IT Miscellaneous Descriptors
drug development
RN 118549-37-4 (glucagon-like peptide-1(7-36)-amide)
50-99-7Q (glucose)
58367-01-4Q (glucose)
9004-10-8 (insulin)

L34 ANSWER 6 OF 9 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on STN
DUPLICATE 1
ACCESSION NUMBER: 2001:506591 BIOSIS <<LOGINID::20070124>>
DOCUMENT NUMBER: PREV200100506591
TITLE: Urinary excretion of glucagon-like peptide 1 (GLP -1) 7-36 amide in human type 2 (non-insulin-dependent) diabetes mellitus.
AUTHOR(S): Lugari, R. [Reprint author]; Ugolotti, D.; Dei Cas, A.; Barilli, A. L.; Iotti, M.; Marani, B.; Orlandini, A.; Gnudi, A.; Zandomeneghi, R.
CORPORATE SOURCE: Cattedra di Endocrinologia, Via Gramsci 14, 43100, Parma, Italy
endoparm@ipruniv.cce.unipr.it
SOURCE: Hormone and Metabolic Research, (September, 2001) Vol. 33, No. 9, pp. 568-571. print.
CODEN: HMMRA2. ISSN: 0018-5043.
DOCUMENT TYPE: Article
LANGUAGE: English
ENTRY DATE: Entered STN: 31 Oct 2001
Last Updated on STN: 23 Feb 2002
AN 2001:506591 BIOSIS <<LOGINID::20070124>>
DN PREV200100506591
TI Urinary excretion of glucagon-like peptide 1 (GLP-1) 7-36 amide in human type 2 (non-insulin-dependent) diabetes mellitus.
AU Lugari, R. [Reprint author]; Ugolotti, D.; Dei Cas, A.; Barilli, A. L.; Iotti, M.; Marani, B.; Orlandini, A.; Gnudi, A.; Zandomeneghi, R.
CS Cattedra di Endocrinologia, Via Gramsci 14, 43100, Parma, Italy
endoparm@ipruniv.cce.unipr.it
SO Hormone and Metabolic Research, (September, 2001) Vol. 33, No. 9, pp. 568-571. print.
CODEN: HMMRA2. ISSN: 0018-5043.
DT Article
LA English
ED Entered STN: 31 Oct 2001
Last Updated on STN: 23 Feb 2002
AB The urinary excretion of insulinotropic glucagon-like peptide 1 (GLP-1) was investigated as an indicator of renal tubular integrity in 10 healthy subjects and in 3 groups of type 2 diabetic patients with different degrees of urinary albumin excretion rate. No significant difference emerged between the groups with respect to age of the patients, known duration of diabetes, metabolic control, BMI, or residual beta-cell pancreatic function. Endogenous creatinine clearance was significantly reduced under conditions of overt diabetic nephropathy, compared with normo and microalbuminuric patients (p<0.01). Urinary excretion of GLP-1 was significantly higher in normoalbuminuric patients compared to controls (490.4+-211.5 vs. 275.5+-132.1 pg/min; p<0.05), with further increase under incipient diabetic nephropathy conditions (648.6+-305 pg/min; p<0.01). No significant difference resulted, in contrast, between macroproteinuric patients and non-diabetic subjects. Taking all patients examined into account, a significant positive relationship emerged between urinary GLP-1 and creatinine clearance (p=0.004). In conclusion, an early tubular impairment in type 2 diabetes would occur before the onset of glomerular permeability alterations. The tubular dysfunction seems to evolve with the development of persistent microalbuminuria. Finally, the advanced tubular involvement, in terms of urinary GLP1 excretion, under overt diabetic nephropathy conditions would be masked by severe concomitant glomerular damage with the coexistence of both alterations resulting in a peptide excretion similar to control subjects.
CC Biochemistry studies - Proteins, peptides and amino acids 10064
Metabolism - Metabolic disorders 13020
Urinary system - Pathology 15506
Endocrine - General 17002
Endocrine - Pancreas 17008
IT Major Concepts
Clinical Endocrinology (Human Medicine, Medical Sciences); Nephrology (Human Medicine, Medical Sciences)
IT Diseases
diabetic nephropathy: endocrine disease/pancreas, metabolic disease, urologic disease
Diabetic Nephropathies (MeSH)
IT Diseases
type 2 diabetes mellitus: endocrine disease/pancreas, metabolic disease, non-insulin-dependent diabetes mellitus
Diabetes Mellitus, Non-Insulin-Dependent (MeSH)
IT Chemicals & Biochemicals
creatinine; glucagon-like peptide 1: renal tubular integrity indicator; glucagon-like peptide 1 7-36 amide [GLP-1 7-36 amide]: urinary excretion
IT Miscellaneous Descriptors
glomerular permeability
ORGN Classifier
Hominidae 86215
Super Taxa
Primates; Mammalia; Vertebrata; Chordata; Animalia
Organism Name
human: patient
Taxa Notes
. Animals, Chordates, Humans, Mammals, Primates, Vertebrates

RN 60-27-5 (creatinine)
ANSWER 7 OF 9 EMBASE COPYRIGHT (c) 2007 Elsevier B.V. All rights reserved on STN
ACCESSION NUMBER: 96034762 EMBASE <<LOGINID::20070124>>
DOCUMENT NUMBER: 1996034762
TITLE: Gastric emptying, glucose responses, and insulin secretion after a liquid test meal: Effects of exogenous glucagon-like peptide-1 (GLP-1)-(7-36) amide in type 2 (noninsulin-dependent) diabetic patients.
AUTHOR: Willms B.; Werner J.; Holst J.J.; Orskov C.; Creutzfeldt W.; Nauck M.A.
CORPORATE SOURCE: Department of Medicine, Ruhr-University Bochum, Knappschafts-Krankenhaus, In der Schornau 23-25,44892 Bochum, Germany
SOURCE: Journal of Clinical Endocrinology and Metabolism, (1996) Vol. 81, No. 1, pp. 327-332. .
ISSN: 0021-972X CODEN: JCEMAZ
COUNTRY: United States
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 003 Endocrinology
037 Drug Literature Index
LANGUAGE: English
SUMMARY LANGUAGE: English
ENTRY DATE: Entered STN: 20 Feb 1996
Last Updated on STN: 20 Feb 1996
AN 96034762 EMBASE <<LOGINID::20070124>>
DN 1996034762
TI Gastric emptying, glucose responses, and insulin secretion after a liquid test meal: Effects of exogenous glucagon-like peptide-1 (GLP-1)-(7-36) amide in type 2 (noninsulin-dependent) diabetic patients.
AU Willms B.; Werner J.; Holst J.J.; Orskov C.; Creutzfeldt W.; Nauck M.A.
CS Department of Medicine, Ruhr-University Bochum, Knappschafts-Krankenhaus, In der Schornau 23-25,44892 Bochum, Germany
SO Journal of Clinical Endocrinology and Metabolism, (1996) Vol. 81, No. 1, pp. 327-332. .
ISSN: 0021-972X CODEN: JCEMAZ
CY United States
DT Journal; Article
FS 003 Endocrinology
037 Drug Literature Index
LA English
SL English
ED Entered STN: 20 Feb 1996
Last Updated on STN: 20 Feb 1996
AB The aim of the study was to investigate whether inhibition of gastric emptying of meals plays a role in the mechanism of the blood glucose-lowering action of glucagon-like peptide-1-(7-36) amide [GLP-1-(7-36) amide] in type 2 diabetes. Eight poorly controlled type 2 diabetic patients (age, 58 ± 6 yr; body mass index, 30.0 ± 5.2 kg/m2; hemoglobin A(1c), 10.5 ± 1.2%) were studied in the fasting state (plasma glucose, 11.1 ± 1.1 mmol/L). A liquid meal of 400 mL containing 8% amino acids and 50 g sucrose (327 Kcal) was administered at time zero by a nasogastric tube. Gastric volume was determined by a dye dilution technique using phenol red. In randomized order, GLP-1-(7-36) amide (1.2 pmol/kg · min; Saxon Biochemicals) or placebo (0.9% NaCl with 1% human serum albumin) was infused between -30 and 240 min. In the control experiment, gastric emptying was completed within 120 min, and plasma glucose, insulin, C-peptide, GLP-1-(7-36) amide, and glucagon concentrations transiently increased. With exogenous GLP-1-(7-36) amide (plasma level, .apprx.70 pmol/L), gastric volume remained constant over the period it was measured (120 min; P < 0.0001 vs. placebo), and plasma glucose fell to normal fasting values (5.4 ± 0.7 mmol/L) within 3-4 h, whereas insulin was stimulated in most, but not all, patients, and glucagon remained at the basal level or was slightly suppressed. In conclusion, GLP-1-(7-36) amide inhibits gastric emptying in type 2 diabetic patients. Together with the stimulation of insulin and the inhibition of glucagon secretion, this effect probably contributes to the blood glucose-lowering action of GLP-1-(7-36) amide in type 2-diabetic patients when studied after meal ingestion. At the degree observed, inhibition of gastric emptying, however, must be overcome by tachyphylaxis, reduction in dose, or pharmacological interventions so as not to interfere with the therapeutic use of GLP-1-(7-36) amide in type 2 diabetic patients.
CT Medical Descriptors:
*insulin release
*non insulin dependent diabetes mellitus: DT, drug therapy
*non insulin dependent diabetes mellitus: TH, therapy
*stomach emptying
adult
aged
article
clinical article
clinical trial
controlled study
diabetic angiopathy: CO, complication
diabetic diet
diabetic nephropathy: CO, complication
diabetic neuropathy: CO, complication
diabetic retinopathy

drug effect
drug mechanism
female
glucagon release
glucose blood level
hormone inhibition
human
hypertension: DT, drug therapy
intravenous drug administration
male
postprandial state
priority journal
randomized controlled trial
Drug Descriptors:
*glucagon like peptide 1 [7-36] amide: CM, drug comparison
*glucagon like peptide 1 [7-36] amide: DT, drug therapy
*glucagon like peptide 1 [7-36] amide: PD, pharmacology
*glucagon like peptide 1 [7-36] amide: CT, clinical trial
*glucose: EC, endogenous compound
*insulin: EC, endogenous compound
acarbose: DT, drug therapy
captopril plus hydrochlorothiazide: DT, drug therapy
glibenclamide: DT, drug therapy
isosorbide dinitrate: DT, drug therapy
metformin: DT, drug therapy
metoprolol: DT, drug therapy
nifedipine: DT, drug therapy
placebo: CM, drug comparison
RN (glucagon like peptide 1 [7-36] amide) 119637-73-9; (glucose) 50-99-7, 84778-64-3; (insulin) 9004-10-8; (acarbose) 56180-94-0; (glibenclamide) 10238-21-8; (isosorbide dinitrate) 87-33-2; (metformin) 1115-70-4, 657-24-9; (metoprolol) 37350-58-6; (nifedipine) 21829-25-4
CO Saxon (Germany)

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ACCESSION NUMBER: 93286381 EMBASE <<LOGINID::20070124>>
DOCUMENT NUMBER: 1993286381
TITLE: Preserved incretin effect in type 1 diabetic patients with end-stage nephropathy treated by combined heterotopic pancreas and kidney transplantation.
AUTHOR: Nauck M.A.; Busing M.; Orskov C.; Siegel E.G.; Talartschik J.; Baartz A.; Baartz T.; Hopt U.T.; Becker H.-D.; Creutzfeldt W.
CORPORATE SOURCE: Div. of Gastroenterol./Endocrinology, Department of Medicine, Georg August University, Robert-Koch-Strasse 40,W-3400 Gottingen, Germany
SOURCE: Acta Diabetologica, (1993) Vol. 30, No. 1, pp. 39-45. .
ISSN: 0940-5429 CODEN: ACDAEZ
COUNTRY: Germany
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 003 Endocrinology
006 Internal Medicine
029 Clinical Biochemistry
LANGUAGE: English
SUMMARY LANGUAGE: English
ENTRY DATE: Entered STN: 31 Oct 1993
Last Updated on STN: 31 Oct 1993
AN 93286381 EMBASE <<LOGINID::20070124>>
DN 1993286381
TI Preserved incretin effect in type 1 diabetic patients with end-stage nephropathy treated by combined heterotopic pancreas and kidney transplantation.
AU Nauck M.A.; Busing M.; Orskov C.; Siegel E.G.; Talartschik J.; Baartz A.; Baartz T.; Hopt U.T.; Becker H.-D.; Creutzfeldt W.
CS Div. of Gastroenterol./Endocrinology, Department of Medicine, Georg August University, Robert-Koch-Strasse 40,W-3400 Gottingen, Germany
SO Acta Diabetologica, (1993) Vol. 30, No. 1, pp. 39-45. .
ISSN: 0940-5429 CODEN: ACDAEZ
CY Germany
DT Journal; Article
FS 003 Endocrinology
006 Internal Medicine
029 Clinical Biochemistry
LA English
SL English
ED Entered STN: 31 Oct 1993
Last Updated on STN: 31 Oct 1993
AB Insulin secretion is stimulated better by oral than by intravenous glucose (incretin effect). The contribution of the autonomic nervous system to the incretin effect after oral glucose in humans is unclear. We therefore examined nine type 1 diabetic (insulin-dependent) patients with end-stage nephropathy, studied after combined heterotopic pancreas and kidney transplantation, and 7 non-diabetic kidney recipients (matched for creatinine clearance and immunosuppressive medication). The release of gastric inhibitory polypeptide (GIP) and glucagon-like peptide 1 (GLP-1) immunoreactivity and B cell secretory responses (IR insulin and C-peptide) to oral (50 g) and 'isoglycaemic' intravenous glucose (identical glycaemic profile)were measured by radioimmunoassay. The difference in B cell responses between the two tests represents the contribution of the enteroinsular axis to the response after oral glucose (incretin effect). Insulin responses after the oral glucose challenge were similar in the two patient groups despite systemic venous drainage of the pancreas graft in the pancreas-kidney-transplanted group. In both groups GIP and GLP-1 increased after oral but not after intravenous glucose, and B cell secretory responses were significantly smaller (by 55.2 ± 7.7% and 46.5 ± 12.5%, respectively) with 'isoglycaemic' intravenous glucose infusions. The lack of reduction in the incretin effect in pancreas-kidney-transplanted patients, whose functioning pancreas is denervated, indicates a lesser role for the nervous system and a more important contribution of circulating incretin hormones in mediating the enteroinsular axis in man.
CT Medical Descriptors:
*diabetic nephropathy: SU, surgery
*insulin dependent diabetes mellitus
*kidney transplantation
*pancreas transplantation
adult
article
clinical article
controlled study
female
human
male
Drug Descriptors:
*gastric inhibitory polypeptide: EC, endogenous compound
*glucagon like peptide 1: EC, endogenous compound
*glucose
*insulin: EC, endogenous compound
RN (gastric inhibitory polypeptide) 59392-49-3; (glucagon like peptide 1) 89750-14-1; (glucose) 50-99-7, 84778-64-3; (insulin) 9004-10-8

L34 ANSWER 9 OF 9 MEDLINE on STN
ACCESSION NUMBER: 92288534 MEDLINE <<LOGINID::20070124>>
DOCUMENT NUMBER: PubMed ID: 1600330
TITLE: Basal and nutrient-stimulated pancreatic and gastrointestinal hormone concentrations in type-1-diabetic patients after successful combined pancreas and kidney transplantation.
AUTHOR: Nauck M A; Busing M; Orskov C; Siegel E G; Talartschik J; Baartz A; Baartz T; Holzer H; Hopt U T; Ebert R; +
CORPORATE SOURCE: Abteilung Gastroenterologie und Endokrinologie, Georg-August-Universitat, Gottingen.
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ENTRY DATE: Entered STN: 24 Jul 1992
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AB The secretion of pancreatic and gastrointestinal hormones in the basal state and after nutrient stimuli (50 g glucose, 50 g protein, or 30 g triglyceride administered on separate occasions) was assessed in ten previously type-1-diabetic patients after successful combined kidney and pancreas transplantation (systemic venous drainage). Fasting values were compared to matched non-diabetic kidney-transplanted patients and related to kidney function (endogenous creatinine clearance) and to the type and dosage of immunosuppressive medication. In the fasting state, only IR insulin concentrations were higher in pancreas-kidney-transplanted patients (by 88%; P = 0.001) than in the kidney graft recipients. There were significant inverse correlations of plasma C-peptide, GIP, and gastrin immunoreactivity to endogenous creatinine clearance (kidney function). In response to nutrients, insulin secretion (IR insulin, C-peptide) was significantly stimulated by glucose, and - to a lesser degree - also by protein. Pancreatic glucagon was suppressed by glucose and stimulated by protein ingestion. GIP was raised after glucose and triglyceride more than after protein (P = 0.0003). GLP-1 immunoreactivity was stimulated by all nutrients, with a tendency towards higher responses to protein and fat (P = 0.06). Gastrin was mainly raised by protein. In conclusion, the overall pattern of

pancreatic and gastrointestinal hormone release is normal in patients after combined pancreas-kidney-transplantation, but there are some peculiarities due to (a) systemic venous drainage of the pancreas graft (elevated fasting IR insulin) and (b) impaired kidney function (negative correlation of fasting plasma values to endogenous creatinine clearance for C-peptide, GIP, and gastrin).(ABSTRACT TRUNCATED AT 250 WORDS)

CT Check Tags: Female; Male
Adult
Blood Glucose: ME, metabolism
Diabetes Mellitus, Type 1: BL, blood
*Diabetes Mellitus, Type 1: SU, surgery
Diabetic Nephropathies: BL, blood
*Diabetic Nephropathies: SU, surgery
*Gastrointestinal Hormones: BL, blood
Humans
Kidney Function Tests
*Kidney Transplantation: PH, physiology
Middle Aged
*Pancreas Transplantation: PH, physiology
Pancreatic Function Tests
*Pancreatic Hormones: BL, blood
Research Support, Non-U.S. Gov't

CN 0 (Blood Glucose); 0 (Gastrointestinal Hormones); 0 (Pancreatic Hormones)